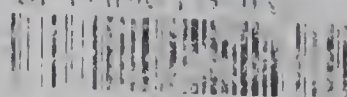


OF TRI-MYSON



2542

to the institution

SCORE

18 5-85

99/10

1. < a



5219

17/11/19



INFANT NUTRITION

INFANT NUTRITION

A Textbook of Infant Feeding for Students and
Practitioners of Medicine

BY

P. C. JEANS, A.B., M.D.

Professor of Pediatrics, College of Medicine
State University of Iowa, Iowa City

AND

WILLIAMS McKIM MARRIOTT, B.S., M.D.

Late Professor of Pediatrics, Washington University School of Medicine;
Physician in Chief, St. Louis Children's Hospital, St. Louis

FOURTH EDITION



ST. LOUIS
THE C. V. MOSBY COMPANY

1947

2540

COPYRIGHT, 1930, 1935, 1941, 1947, BY THE C. V. MOSBY COMPANY

(All rights reserved)

Reprinted February, 1948

Printed in the
United States of America

L-90,32

N 47

CFTRI-MYSORE



2540

Infant nutrition.

*Press of
The C. V. Mosby Company
St. Louis*

PREFACE TO FOURTH EDITION

The science of nutrition has advanced greatly since publication of the preceding edition. This advancement includes many phases applicable to infant nutrition. Some of the important new information applies specifically to infants. Because of increased knowledge numerous changes and additions have been made in this edition. All parts of the book have been revised and many sections have been rewritten. New illustrations have been provided, particularly for the chapter on techniques. The purpose of the fourth edition remains the same as it was originally. The book is intended to be useful to both the student and the practitioner.

P. C. J.

PREFACE TO THIRD EDITION

The important contribution of Marriott to infant nutrition consisted in the replacement of empiricisms with scientific fundamentals and the establishment of the entire subject on a simpler, more logical and more common-sense basis. For this accomplishment he took no special credit to himself. In the preface to the first edition of this book he stated that the purpose of the book was to summarize the present-day knowledge of the nutrition of infants. Since the book was first published, research in nutrition has been extensive. The advances in our knowledge made necessary a second edition after a few years. Again a new edition has become desirable for the same reason. Also it becomes necessary that the name of another author appear on the title page. The present editor was associated with the former author for a period of years and no fundamental differences exist in the two points of view. In this third edition the purpose of the

book remains the same, namely, to bring together such facts and ideas as have a practical bearing on infant nutrition and to present them in such a way as to make them useful to the practitioner and student.

P. C. J.

Iowa City, Iowa.

PREFACE TO FIRST EDITION

There is perhaps no field in medical practice in which more difference of opinion has existed than in that of infant feeding. So numerous have been the theories advanced and so diverse the methods of feeding recommended that the practitioner is likely to become hopelessly bewildered. Much of this confusion, however, is avoidable, for the fundamental facts concerning the nutritional requirements of infants are now fairly well established, and the most that any type of feeding can accomplish is to fulfill these requirements.

It is the purpose of this book to summarize present-day knowledge concerning the nutritional requirements of infants under normal and pathological conditions and to indicate the effects of failure to meet any or all of these requirements.

In order that the results of feeding on the growth and development of infants may properly be appraised, a knowledge of normal growth and development is essential—a chapter on this subject has accordingly been included.

A food, although a complete one, cannot be expected to meet the nutritional requirements unless it is one which is capable of utilization by the infant; in other words, it must be digestible and absorbable and not capable of causing injury to the body. The selection of such a food must be based on an understanding of the characteristics of the chief food elements and the processes of digestion and absorption in infancy as well as alterations of these processes which are likely to occur under abnormal conditions.

The chapters on Metabolism and Digestion outline the present-day viewpoint on these subjects.

With a thorough knowledge of the nutritional needs of the infant and of the means by which it is possible to meet those needs, the practitioner is in a position to appraise the value of any given type of feeding. If a formula or type of food is one which meets fully the requirements and at the same time is one which is capable of utilization without harm to the body, it will be successful. If it fails to meet any one of the essential requirements, it will fail as a whole. If these self-evident facts were more fully appreciated, there would be fewer failures due to the feeding of well-balanced digestible formulas in insufficient amounts to meet the minimal energy requirements or the feeding of mixtures meeting the caloric demands but deficient in such essential elements as protein or some of the vitamins.

There are numerous ways in which infants may be fed successfully, and there is no one method of feeding or type of food which is to be recommended to the exclusion of others; on the other hand, there is no reason to resort to the use of complicated formulas or expensive proprietary preparations when the particular requirements in the individual case may be fully met by simpler means. The simpler means are especially stressed in this book, as any complication of methods is not only unnecessary but introduces greater chances for error. Sufficient information is given, however, it is hoped, to enable the practitioner to use intelligently such special foods as are now available or may from time to time be introduced.

The chief gastrointestinal disturbances of infants—vomiting, diarrhea and constipation—receive especially full consideration, for the occurrence of these conditions may wreck any carefully laid plan of feeding.

So important is the relationship of infection to nutrition that a separate chapter is devoted to the subject. It is becoming recognized that many of the disturbances of infants which have previously been attributed to dietary

faults are in reality the result of infections. The relationship, however, is a reciprocal one, for infections often occur because the nutrition has been inadequate. An infant suffering from an unrecognized otitis media cannot be made to thrive by merely changing the feeding formula, nor on the other hand can an infant with a chronic infection be expected to overcome the infection unless the diet is an adequate one.

Throughout the book an effort has been made to stress the importance of the chemical pathology of the body as related to feeding and nutrition, as an understanding of this enables one to apply much more intelligently therapeutic procedures. This phase of the subject receives especial consideration in the chapters on Anhydremia, Acidosis and Alkalosis, Rickets, and Tetany.

The author believes a thorough exposition of the fundamental principles of nutrition to be of greater value to the practitioner than a mere outline of methods of procedure, for, with a conception of the underlying principles, he can intelligently meet any condition as it arises and be independent of "rule-of-the-thumb" procedures. Nevertheless the practical application of the scientific principles laid down can perhaps best be appreciated by the use of specific examples. For that reason numerous clinical protocols have been introduced illustrating the more common nutritional problems; details of the management of cases described are given. An attempt has thus been made to give the *how* as well as the *why*.

As a further aid from the practical standpoint, separate chapters on Therapeutic Procedures and on Medication in Infancy have been included.

The author makes no claim of originality for the ideas expressed in this book. The successful methods of modern infant feeding are the outcome of the labors of many men in many countries, each contributing some useful or important observation. The last word has not yet been spoken. The establishment of a new fact may, at any

time, change accepted methods of practice. An effort has been made in this book to evaluate present-day knowledge of nutrition on the basis of actual clinical trial, and conclusions have been arrived at not in the laboratory or easy-chair, but in the clinic.

The author takes pleasure in expressing his indebtedness to the various members of the Department of Pediatrics of the Washington University School of Medicine who have contributed many helpful suggestions, and especially to Doctors Theodore C. Hempelmann and Alexis F. Hartmann, who have read the entire manuscript and have made many valuable suggestions.

W. McK. M.

CONTENTS

CHAPTER I		PAGE
GROWTH AND DEVELOPMENT - - - - -		17
Relative Body Proportions During Infancy, 19; Body Length, 20; Weight, 22; Growth of Prematurely Born Infants, 24; Head, 24; Teeth, 26; Chest, Lungs, and Heart, 27; Stomach and Intestinal Tract, 28; Liver, 29; Urinary Tract, 30; Skeleton (Bone), 30; Skin, Hair, and Subcutaneous Tissues, 32; Lymphatic System, 32; The Nervous System, 33; The Special Senses, 35; Body Temperature, 36; Skeletal Muscle and Motor Behavior, 36; Blood, 38; Endocrine Glands, 41.		
CHAPTER II		
ENERGY METABOLISM - - - - -		43
The Significance of Calories, 43; Basal Metabolism, 45; Allowance for Activity, 47; Allowance for Growth, 47; Allowance for Unutilized Food, 48; The Total Energy Requirements, 48.		
CHAPTER III		
PROTEIN METABOLISM - - - - -		52
The Composition of Protein, 52; The Protein Requirement of Infants, 53; Protein Under- and Overnutrition, 55; Proteins Other Than Those of Milk, 56.		
CHAPTER IV		
CARBOHYDRATE METABOLISM - - - - -		58
Lactose (Milk Sugar), 62; Sucrose, 63; Products of Starch Hydrolysis ("Malt Sugars"), 63; Dextrin, 64; Maltose, 65; Dextrose (d-Glucose), 65; Dextrin-Maltose Mixtures, 65; Dextrin-Maltose-Dextrose Mixtures (Corn Syrup), 66; Dextrin-Maltose-Dextrose-Sucrose Mixtures (Flavored Corn Syrup), 66; Mixed Sugars, 67; Molasses, 67; Honey, 68; The Carbohydrates of Fruits, 68; Starch, 68; Sugars as a Cause of Diarrhea, 69; Parenteral Administration of Carbohydrates, 70.		
CHAPTER V		
FAT METABOLISM - - - - -		71
CHAPTER VI		
MINERAL AND WATER METABOLISM - - - - -		77
Inorganic Constituents of the Body, 77; Body Water, Sodium, Potassium and Chloride, 78; Calcium, Phosphorus, and Magnesium, 82; Sulfur, 86; Iron, 87; Copper, 90; Iodine, 91; Fluorine, 92; Manganese, 93; Cobalt, 93; Zinc, 93; Acid-Base Balance, 94.		

CHAPTER VII

	PAGE
THE VITAMINS - - - - -	95
Vitamin A, 96; Vitamin D, 102; Vitamin K, 106; Vitamin E, 109;	
The Vitamin-B Complex, 110; Thiamine, 111; Riboflavin, 115;	
Nicotinic Acid, 117; Folic Acid, 119; Pyridoxine, 120; Biotin, 120;	
Choline, 121; Other B Complex Factors, 121; Ascorbic Acid, 122;	
Vitamin P, 126.	

CHAPTER VIII

SUMMARY OF THE NUTRITIONAL REQUIREMENTS OF INFANTS - - - -	127
Calories, 127; Proteins, 127; Carbohydrates, 128; Fats, 128; Mineral Salts, 129; Water, 129; Vitamins, 129.	

CHAPTER IX

DIGESTION IN INFANCY - - - - -	131
Salivary Digestion, 131; Gastric Digestion, 132; Intestinal Digestion, 137; Bacteriology of the Gastrointestinal Tract, 141.	

CHAPTER X

THE STOOLS IN INFANCY - - - - -	145
Meconium, 145; Stools of the Breast-Fed Infant, 145; Stools of the Artificially Fed Infant, 146; The Number of Stools, 146; The Color of the Stools, 147; Curds, 148; Mucus, 149; Starch, 149; Cellular Elements, 149; Bacteriology of the Stools, 150; Significance of Stool Examination, 151.	

CHAPTER XI

BREAST FEEDING OF THE NORMAL INFANT - - - - -	152
General Considerations, 152; Contraindications to Breast Feeding, 154; Characteristics of Human Milk, 156; Colostrum, 156; Composition of Human Milk, 156; The Hygiene of the Nursing Mother, 161; Amounts of Milk Secreted, 161; Diet of the Nursing Mother, 162; Care of the Breasts and Nipples, 165; Technique of Breast Feeding, 166; Characteristics of the Normal Breast-Fed Infant, 171; Underfeeding, 172; Overfeeding, 173; Unsuitable Milk, 174; Gastrointestinal Disturbances of Breast-Fed Infants, 175; Mixed Feedings, 175; Additions to the Diet of the Breast-Fed Infant, 176; Weaning, 177; Wet Nursing, 178; Manual or Mechanical Expression of Milk From the Breast, 180.	

CHAPTER XII

ARTIFICIAL FEEDING - - - - -	182
General Considerations, 182; The Requirements of a Satisfactory Artificial Feeding, 185; Bacterial Contamination of the Infant's Food, 187; Chemical Contamination of the Formula Diluent (Well Water Cyanosis), 188; Digestibility of Milk Formulas, 188; Preference of the Infant for Various Types of Formulas, 190.	

CHAPTER XIII

PAGE

COMPOSITION AND CHARACTER OF COW'S MILK - - - - -	191
The Enzymes of Milk, 193; Miscellaneous Constituents of Milk, 193; Bacteriology of Cow's Milk, 194; Methods for Reduction of the Bacterial Count of Milk, 196; Certified Milk, 197; Pasteurized Milk, 198; Boiled Milk, 199; Soft-Curd Milks, 199; Vitamin-D Milk, 201; Modification of Milk for Infant Feeding, 201.	

CHAPTER XIV

FEEDING THE NORMAL INFANT WITH WHOLE SWEET MILK MIXTURES - -	203
Construction of the Formula, 205; Technique of Preparing the Formula, 209; The Nursing Bottle, 211; Technique of Feeding, 212.	

CHAPTER XV

EVAPORATED MILK MIXTURES - - - - -	213
Characteristics of Evaporated Milk, 213; Construction of Formulas With Evaporated Milk, 215; Technique of Preparing the Formula, 217.	

CHAPTER XVI

ACID MILK - - - - -	218
Lactic Acid Milk, 220; Bacterially Soured Milk, 221; Milk Soured by the Addition of Lactic Acid, 222; Citric Acid Milk, 224; Other Acid Milks, 225; A Standard Acid-Milk Formula, 225; Acidified Evaporated Milk Formulas, 227.	

CHAPTER XVII

SPECIAL AND PROPRIETARY FOODS - - - - -	229
Goat's Milk, 229; Dried Milk, 230; Carbohydrate Preparations, 232; Condensed Milk, 232; Malted Milk, 233; Milk-Carbohydrate Mixtures Prepared Exclusively for Infant Feeding, 234; Foods Designed to Meet Special Conditions, 235; Protein Milk (Albumin Milk, "Eiweiss Milch"), 235; Hypoallergenic Milks, 236; Milk Substitutes, 237.	

CHAPTER XVIII

THE DIET OF THE NORMAL INFANT - - - - -	238
Milk, 238; Cod-Liver Oil, 238; Orange Juice and Tomato Juice, 239; Egg, 240; Vegetables, 242; Fruit, 243; Cereal, 243; Meat, 245; Other Foods, 246; General Discussion, 248.	

CHAPTER XIX

MALNUTRITION (ATHREPSIA, MARASMUS) - - - - -	251
The Causes of Malnutrition, 251; Underfeeding, 252; Deficient Utilization of Food, 254; Effects of Infection, 254; Effects of Congenital Anomalies, 255; Other Causes, 256; The Symptomatology and Pathology of Undernutrition, 257; The Treatment of Malnutrition, 261.	

CHAPTER XX	
DIARRHEA - - - - -	PAGE 270
General Considerations, 270; Symptoms of Diarrhea, 278; The Effects of Diarrhea on the Body, 280; Diminished Absorption of Food, 280; Anhydremia, 281; Acidosis, 284; Toxemia From Intestinal Bacteria, 285; The Differential Diagnosis of Diarrhea, 285; Prophylaxis of Diarrhea, 288; Treatment of Diarrhea, 290; General Principles, 290; Treatment of Diarrhea in the Breast-Fed Infant, 293; The Treatment of Mild Diarrhea in Artificially Fed Infants, 295; Treatment of the Severe Forms of Diarrhea (Alimentary Intoxication, Toxicosis, Cholera Infantum, Anhydremia), 298; Medicinal Treatment, 309.	
CHAPTER XXI	
BACILLARY DYSENTERY (ILEOCOLITIS, INFECTIOUS DIARRHEA) - - - -	312
Pathology, 312; Symptoms, 314; Complications, 316; Prognosis, 317; Treatment, 318; Chemotherapy, 321.	
CHAPTER XXII	
THE CELIAC SYNDROME - - - - -	323
Celiac Disease, 323; Etiology and Pathology, 324; Symptoms, 325; Treatment, 326; Cystic Fibrosis of the Pancreas, 330.	
CHAPTER XXIII	
VOMITING - - - - -	333
Vomiting Due to Swallowing of Air, 333; Overdistention of the Stomach by Too Frequent or Too Large Feedings, 334; Vomiting Due to Unsuitable Composition of the Food, 335; Vomiting Due to Improper Clothing and Handling, 336; Vomiting Due to Parenteral Infections, 336; Habit or "Nervous" Vomiting; Rumination, 337; Gastroenterospasm, 338; Obstruction of the Gastrointestinal Tract, 338; Atresia of the Esophagus, 339; Pyloric Stenosis, 340; Obstruction of the Duodenum, 349; Intussusception, 350; Anhydremia, 352; Allergy, 352; Intracranial Conditions, 352; Toxic States, 352; The Effects of Vomiting on the Body, 353.	
CHAPTER XXIV	
COLIC, FLATULENCE, AND GASTROENTEROSPASM - - - - -	356
Treatment, 358.	
CHAPTER XXV	
CONSTIPATION - - - - -	361
Symptoms, 363; Treatment, 363.	
CHAPTER XXVI	
ALLERGY - - - - -	368

CHAPTER XXVII

PAGE

PREMATURITY - - - - -	375
Prematurity and the Newborn Period, 375; The Management of the Prematurely Born Infant, 381.	

CHAPTER XXVIII

COMMON INFECTIONS WHICH ARE ASSOCIATED WITH NUTRITIONAL DISTURBANCES - - - - -	393
Rhinopharyngitis, 393; Otitis Media, 394; Mastoiditis, 397; Sinusitis, 402; Pyelitis, 403; Tuberculosis, 406; Syphilis, 407.	

CHAPTER XXIX

RICKETS - - - - -	411
Etiology, 411; Pathogenesis and Pathology, 417; Symptoms, 424; Diagnosis, 431; Prevention and Treatment, 432.	

CHAPTER XXX

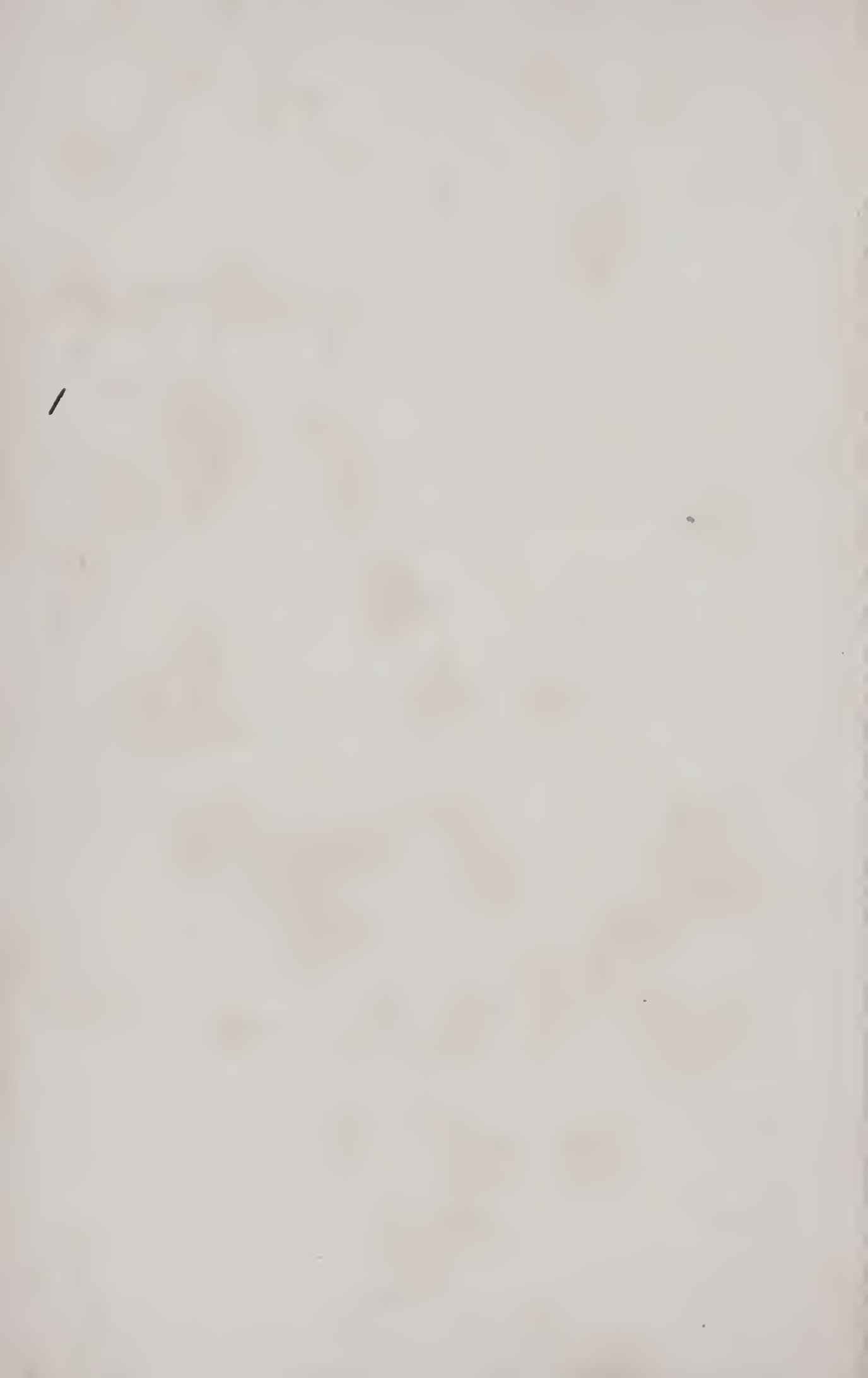
TETANY (SPASMOPHILIA) - - - - -	437
Etiology and Pathogenesis, 437; Symptoms, 440; Diagnosis, 443; Treatment, 445.	

CHAPTER XXXI

SCURVY (SCORBUTUS) - - - - -	450
Etiology, 450; Pathology, 451; Symptoms, 454; Diagnosis, 455; Treatment, 457.	

CHAPTER XXXII

MISCELLANEOUS TECHNIQUE - - - - -	459
Collection of Urine, 459; Urine Cultures, 461; Stool Cultures, 461; Pinworm Ova, 462; Collection of Blood, 462; Grouping and Matching of Blood for Transfusion, 468; Blood Transfusion, 469; Saline Administration, 481; Subcutaneous Injection, 481; Intraperitoneal Injection, 482; Dextrose (Glucose) Administration, 483; Fluids for Parenteral Use, 484; Continuous Intravenous Injection (Venoclysis), 485; Gavage, 487; History Taking, 488; The Clinical Chart, 490; Laboratory Sheet, 494; Drugs, 494.	



INFANT NUTRITION

CHAPTER I

GROWTH AND DEVELOPMENT

Physical growth may be defined as the increase in the quantity of living tissue of the body, either by increase in the size of cells already present or by the development of new cells. The term development includes both physical growth and the increase in ability of the body to function, both physically and mentally.

Many factors influence the rate of physical growth. Most of them can be classified under heredity and environment. Heredity probably influences the balance between the secretions of the various endocrine glands. These in turn regulate the general pattern of growth, determining the type of body build and probably limiting the maximum size that an individual can attain. Environment is more controllable. Nutrition is by far the most important of the controllable factors that determine the rate of growth of the normal infant. Other factors, as insufficient rest and disease, either acute or chronic, decrease the rate of growth, but these factors exert their influence on growth through their effect on nutrition.

Fully adequate nutrition permits optimum growth. Growth of the infant begins at conception. The fetus is considered as parasitic on the mother and formerly the nutrition of the fetus was considered adequate regardless of the state of the mother's health or nutrition. Studies of the relation between maternal nutrition and the physical status of the infant have proved the fallacy of this concept.

Poor nutrition during pregnancy affects the fetus even more than the mother. Survey studies of the influence of prenatal diet on the mother and child have been made by Ebbs, Tisdall, and Scott in Toronto; by Burke, Harding, and Stuart in Boston; and by Cameron and Graham in Glasgow. The findings of the three groups are similar. Those mothers whose diets were fully adequate maintained better health throughout pregnancy, had fewer complications of labor and were, in general, better obstetrical risks than the mothers whose diets were poor. The incidence of miscarriage, stillbirth and premature birth was much smaller in the well-fed group; their infants were in better physical condition at birth, fewer neonatal deaths occurred, fewer and less severe illnesses were observed up to the age of six months than was the case among infants of poorly fed mothers. In addition more of the well-fed mothers were able to nurse their infants successfully.

The Boston group found in their survey that maternal diets tended to be low in protein and that if the protein intake was adequate, containing 75 grams or more daily, the infants were longer and heavier at birth than infants of mothers whose protein intakes were inadequate. Notwithstanding the increase in size of the infants, complications of labor were fewer.

Studies with animals have shown that poor maternal diets can result in congenital malformations of the fetus. Deficiency of vitamin A results in abnormalities of the eye and skeleton in pigs, rats, and cattle. Harelip, cleft palate, missing limbs, blindness, and missing eyeballs were observed. Maternal deficiency of riboflavin in rats results in skeletal deformities of the offspring, including cleft palate. Deficiency of vitamin D also results in skeletal deformities. If the dietary deficiencies are extreme, the embryos die in utero. No studies have yet been reported of the effect of deficiencies of human maternal diets on the production of congenital malformation in the infant.

Maternal rubella within the first three months of pregnancy is a cause of congenital defects in human infants. Infants born of such pregnancies have exhibited deaf-mutism, cataract, malformations of the heart, microcephaly, and obliteration of the bile ducts. The effect of other toxic disturbances during the period of embryonal growth has not yet been reported.

After birth, the rate of growth and development of the infant depends in large measure on the adequacy of his nutrition. Average figures for the weights and measurements of infants at various ages should not be taken as an absolute guide in determining the development of an individual infant. Such statistics have been gathered from large groups of infants whose chief criterion of normality usually is that they show no illness at the time of measurement. Comparison of recent tables with older studies shows that the average infant of today grows faster, both in length and weight, than the average baby of twenty years ago. Babies of healthy stock, given abundant food of suitable composition, are generally distinctly heavier and larger in all measurements than the accepted averages of the present day.

Relative Body Proportions During Infancy

At birth the relative proportions of the parts of the body differ markedly from those at maturity. In the adult the head forms one-eighth and the legs about one-half of the total body length. In contrast, the head of the newborn infant constitutes one-fourth of the total length of the body, the remainder being divided about equally between trunk and legs. These proportions in length do not change to any great extent during the first year of life. The relative width of the body at the shoulders and hips increases most rapidly during the first three months. The circumference of the head is somewhat greater than that of the chest throughout most of the first year. Only after eighteen months of age does the chest circumference exceed that

of the head. The general type of body build—that is, whether the child is of the linear or the lateral (stocky) type of body configuration—can be ascertained in early infancy.

Body Length

Normal babies born at term may vary 10 cm. (2.5 in.) or more in length, depending probably both on heredity and on nutrition in utero. According to Meredith the mean

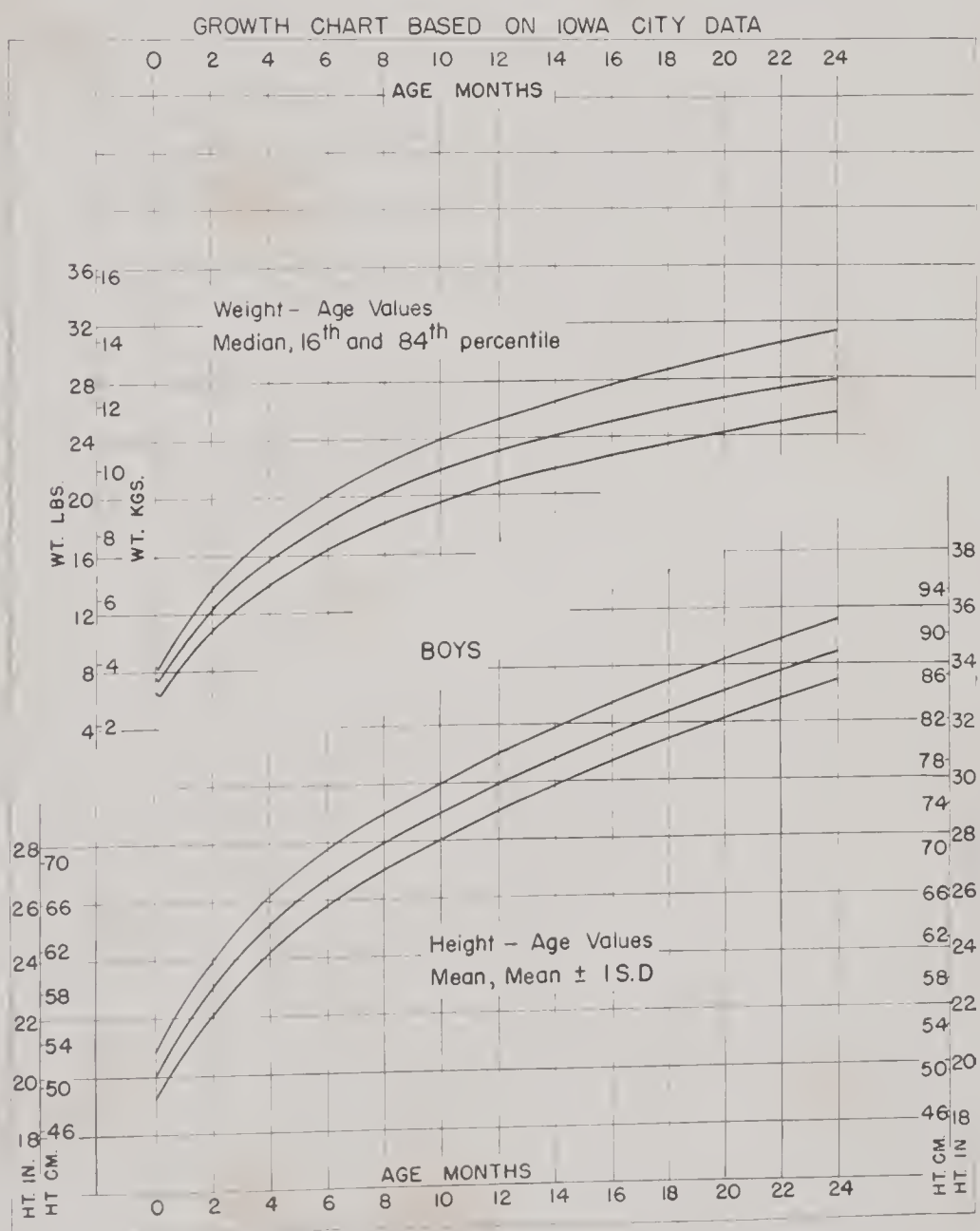


Fig. 1.—Height-age and weight-age chart for boys, birth to two years.

birth length for boy infants of this country is 50.8 cm. (20 in.), for girls, 50.0 cm. (19.6 in.) with a normal range for both of from 45 to 55 cm. (17.7 to 21.6 in.). A slight decrease in length may occur in the neonatal period, due to recovery from the molding and stretching incident to birth. This decrease rarely is more than 1 cm. Growth in length is most rapid during the first three months after birth and becomes progressively slower during later infancy. The average baby grows about 25 cm. (10 in.) during the first

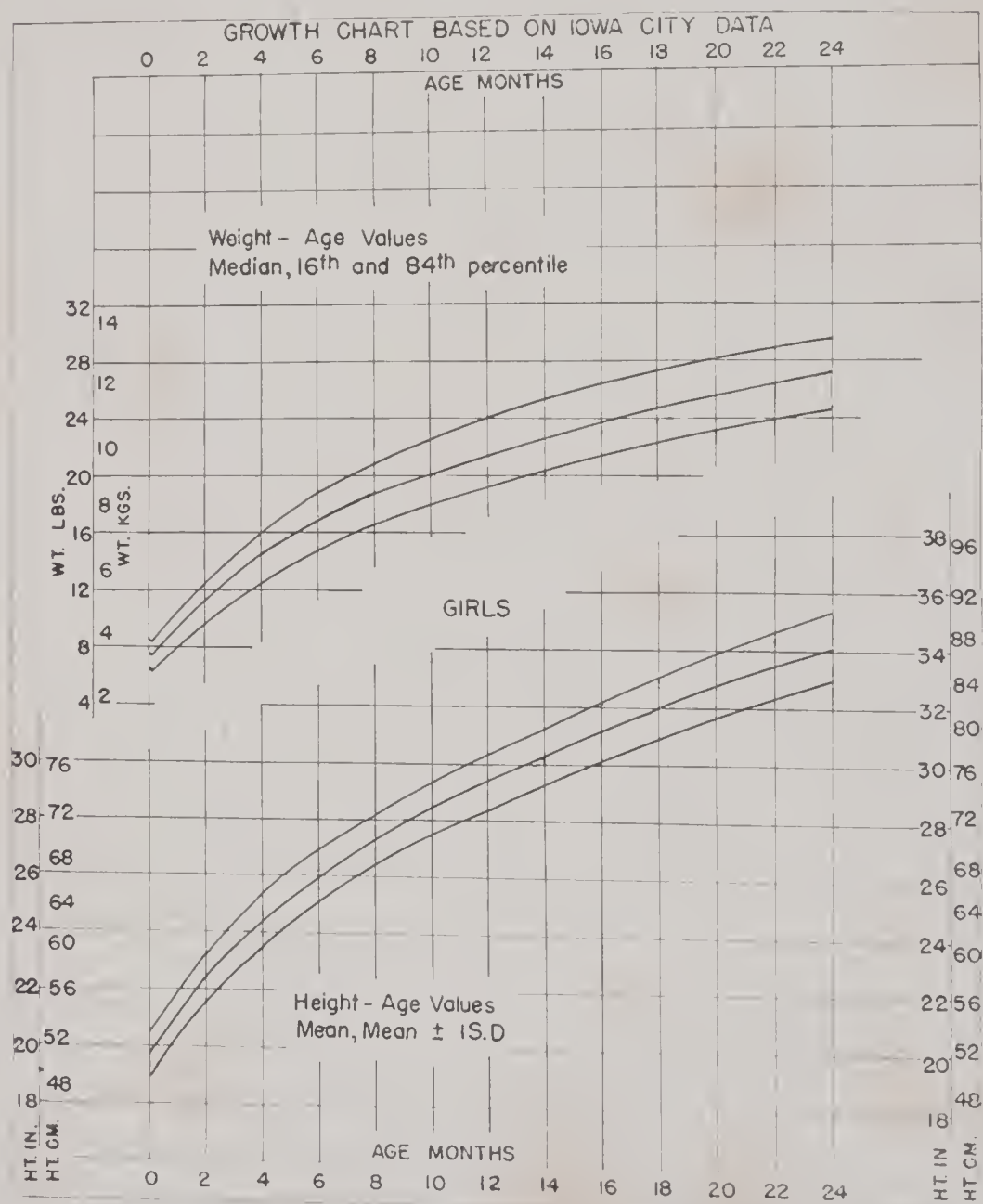


Fig. 2.—Height-age and weight-age chart for girls, birth to two years.

year, divided as follows: 10, 7, 5, and 3 cm. in the first, second, third, and fourth quarters, respectively.

A tall baby apparently does not grow more rapidly than a short one during the first year, but he maintains his lead in stature. During the second year the tall child continues to maintain his lead over his shorter brother. Whatever the birth length, the well-fed baby will usually grow at a rate somewhat greater than the average for his birth length.

In general, girls are about 0.8 cm. shorter at birth than boys and about 2 cm. shorter at a year. Otherwise the general trends of growth are the same in the two sexes.

Growth in length is measured conveniently by placing a table against a wall with the baby on the table, the top of his head against the wall. The knees can be straightened and the length measured on a yardstick kept at right angles to the wall, using a book or short piece of board at the soles of the feet. Greater precision is attained by having some means of maintaining the footboard at a right angle to the measuring stick.

Weight

The weight of a newborn infant will depend on his body size and the amount of fat. It is therefore much more variable than the body length. The average body weight for a boy baby 51 cm. (20 in.) long is about 3400 Gm. (7.5 pounds), with a normal range of from 2700 to 4000 Gm. (6 to 9 pounds), and a maximum normal weight of 5100 Gm. (11.2 pounds). Therefore, a newborn infant who might be anywhere from 46 to 55 cm. (18 to 22 in.) in length, might weigh from 2000 to 5000 Gm. (4.4 to 11 pounds) and still be considered normal. It is thus necessary to relate the infant's weight to his height (length) as well as to his age; the baby can then readily be classified as average, slender, or fat. When weight is related to height, little difference exists in weights of boy and girl infants, that is, girls 55 cm. long weigh practically the same as boys the same length.

Regardless of birth weight, a loss of 200 to 250 Gm. (7 to 8 ounces) occurs during the first week of life. This loss is due in large part to emptying the intestine of material accumulated before birth (meconium). About one-fifth of the weight loss is due to a deficit in the food intake during the first week in relation to the requirement. After the baby receives sufficient food, this lost weight is soon regained, and the well infant will maintain a weight within the normal range for his length throughout the period of infancy.

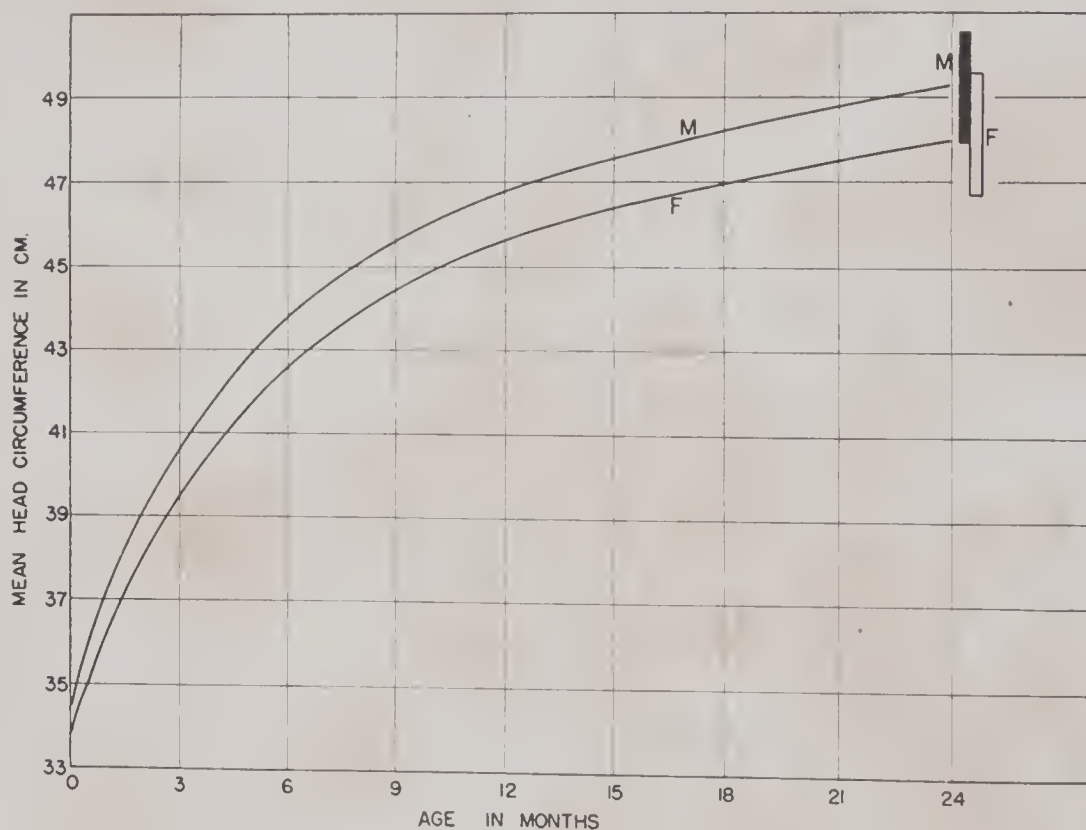


Fig. 3.—Mean head circumference of male and female infants during the first two years after birth. The standard deviation for each sex remains approximately constant during this period at ± 0.85 cm. (0.33 in.) as shown by the bars. (After Meredith.)

While the actual weight of a baby at any given time depends on his length and state of nutrition, regular gain in weight is one of the important criteria of progress. A baby who is given a proper diet and is gaining well is usually thriving in all other respects. Gain in weight, however, if continued for a month or more without accompanying gain

in length, may be an indication of improper feeding. A young baby should be weighed at least twice a week; once a week is sufficient after two months of age. The average healthy infant doubles his birth weight by four or five months of age and triples it by the end of the first year.

Growth of Prematurely Born Infants

Any infant weighing less than 2500 Gm. at birth is usually considered immature, the majority of such babies being those prematurely born, that is, born after less than 270 days of gestation. These infants have a more difficult period of adjustment to extrauterine life than do full-term infants, and often growth is slower in starting. After gain in weight is well established, it is rapid: often the birth weight is doubled at two months and quadrupled at a year. While these percentage gains are greater than those of a full-term infant, the actual weight gain, in pounds or grams, is often less than that attained by the full-term infant. Similarly, growth in length may be 10 cm. (4 in.) or more greater than average during the first year, yet often the prematurely born baby at a year of age is still below average height of full-term infants. Infants born only slightly prematurely may make up their deficiency and be equal to the average by the end of the first year. In general, however, it appears that the infant born several weeks prematurely cannot be expected to "catch up" with his normal siblings in either height or weight until he is two or more years old.

Head

The head of a newborn infant is twice as large in proportion to the remainder of the body as is the head of an adult. The head also grows more rapidly during infancy than at any later time, attaining approximately one-half of its total postnatal increase in circumference during the first year. Like the growth of the body as a whole, growth of the head is most rapid in early infancy and becomes appreciably

slower toward the end of the first year. Head size is determined most simply by the circumference, measured over the greatest prominence of the occiput and of the glabella. Measurements of head circumference immediately after birth may be somewhat misleading because of molding of the head in its passage through the birth canal. The head circumference of boys not only exceeds that of girls at birth but increases at a faster rate during the first six months. At birth the difference in head size between the sexes averages 0.6 cm. (0.25 in.); by six months of age it has increased to 1.2 cm. (0.5 in.). From six months to two years the difference in head size remains constant at 1.2 cm. (0.5 in.). The mean head girth for newborn boys is 34.5 cm. (13.6 in.) and for girls, 33.9 cm. (13.3 in.). At three months of age the mean values for the two sexes are 40.4 and 39.4 cm. (15.8 and 15.5 in.), respectively; at six months 43.7 and 42.5 cm. (17.2 and 16.8 in.); at nine months 45.6 and 44.5 cm. (17.9 and 17.5 in.); at twelve months 46.7 and 45.5 cm. (18.4 and 17.9 in.); and at two years 49.2 and 48 cm. (19.4 and 18.9 in.), respectively. The range of variation among normal infants at all ages is close to 0.85 cm. ($\frac{1}{3}$ in.) on either side of the mean. Growth of the head continues even with marked underfeeding, so that the head of an undernourished baby may appear disproportionately large as compared with the remainder of the body. As the growth of the head is determined by the rate of growth of the brain, if for any reason growth of the brain becomes arrested, the head circumference remains small.

The face is relatively small in infancy; the greater part of the head is occupied by the brain. The cranium or brain case is made up of seven bones of the membranous type. At birth these are all separate. The boundary between any two is called a suture, and the junction of three or more is known as a fontanel. Six fontanels exist at birth, but not more than two of these are normally palpable in a full-term infant. These are both located in the midline and are

designated as anterior and posterior. The posterior fontanel may be closed at birth and normally is closed by the end of the second month. The anterior fontanel remains open for many months. In the normal child it is usually closed by eighteen months; in many robust, well-nourished babies it may be closed at one year or even earlier. Arrested brain development is accompanied by early fontanel closure. Delay in closure is caused most often by those conditions in which calcification is delayed, such as rickets and cretinism.

Teeth

All of the first or deciduous set of teeth begin to develop before birth. Calcification of the first teeth begins in the fourth month of fetal life, the incisors being calcified first. All the deciduous teeth are in process of calcification by the sixth month of fetal life. The process of enamel and dentine formation begins in the crown and gradually extends toward the base of the tooth. The mother's health and nutrition during pregnancy play a considerable role in the efficiency of calcification of the deciduous teeth. The first permanent molar begins to calcify at the time of birth. The deciduous molars are only partly calcified at this time. Calcification of the permanent central incisors, cuspids and upper lateral incisors has begun by six months of age. The permanent upper lateral incisors begin to calcify at about one year of age, the first bicuspid at about two years and the second molars at about two and one-half years. Thus the character of many of the deciduous teeth and all the permanent teeth will depend largely on the nutrition during infancy and early childhood.

The twenty teeth of the first set are not completely erupted until the child is two to two and one-half years old. Usually the first teeth to erupt are the two lower central incisors, then the two upper central incisors and the two upper lateral incisors. These six teeth often are present by nine months of age and should all be erupted by the first

birthday. The lower lateral incisors and the four anterior molars erupt between the ninth and eighteenth months. The four canines ("eye" and "stomach" teeth) and the four posterior molars appear when the child is fifteen to thirty months of age.

Many otherwise normal infants do not conform strictly to the stated schedule. Well-nourished children tend to erupt their teeth somewhat earlier than average; marked delay in dentition may be due to rickets or to thyroid deficiency. The eruption of teeth is a physiologic process and, while it may cause some discomfort and restlessness, should not be associated with any real illness. Occasionally an infant develops loss of appetite and irritability, slight fever, and lessened digestive capacity, so that if his customary diet is continued, diarrhea may develop. The general tendency of mothers to ascribe all illness occurring during this period to "teething" results in much harm, since the true cause of the illness is left undiagnosed and untreated.

Chest, Lungs, and Heart

The circumference of the chest at birth is slightly smaller than that of either the head or the abdomen and averages about 34 cm. (13.5 in.). At about the end of the first year the head, chest, and abdomen are about the same in circumference; thereafter the chest grows more rapidly than either. In prematurely born infants, the disproportion between head and chest is more marked than in normal infants; undernourished infants have the same appearance, as the chest fails to develop as rapidly as the head. The chest cavity of the young infant is somewhat funnel-shaped, being narrow at the top and widening at the diaphragm, while in cross section it is almost circular. As the infant grows, the chest cavity becomes relatively larger at the top, and widens so that the transverse diameter becomes greater than the anterior-posterior.

In the young infant the chest wall is soft and flexible, chiefly because of the large amount of cartilage at the

sternal end of the ribs. At birth the ribs are almost at right angles to the long axis of the body. Little change in angulation occurs during infancy.

The lungs of the newborn infant are characterized by having relatively larger trachea and bronchi, more interstitial tissue, and smaller alveoli than those of adults. Aeration of the lungs may not be complete for several days after birth. Respirations are rapid and irregular, even slight disturbances causing a marked increase in rate. The rate during sleep averages about 40 a minute, but may increase to 60 during excitation. The depth of respiration, as well as the rhythm, is irregular and may be periodic. Regular rhythmic respiration is not established until the end of the second year.

The heart is a little larger in proportion to the body weight at birth than in later life. The size, shape, and position are as variable among infants as among adults. The rate of growth of the heart is negligible for the first three or four months; then its growth rate parallels that of the body as a whole until the end of the first year. The resting contraction rate is rapid, from 120 to 150 to the minute during the first month, and 100 to 140 a minute during the remainder of the year. Like the respiratory rate, the heart rate is characterized by instability, small stimuli producing relatively large increases in rate, even in health and during sleep. It has been observed that the ratio of respiratory rate to pulse rate usually approximates 1:4. The systolic blood pressure is about 80 millimeters of mercury at birth and increases to 95 or 100 by two weeks of age. The circulation rate is about twice as rapid in infants as in adults, the entire round being accomplished in about twelve seconds.

Stomach and Intestinal Tract

The position of the stomach during early infancy is almost transverse. As the infant grows older and assumes the upright position for a portion of the day, the pyloric

end of the stomach moves downward. The physiologic capacity of the stomach is always greater than the anatomic capacity for two reasons: the stomach is capable of considerable distention and some food leaves the stomach while the infant is still eating. The physical capacity of the stomach is indicated by the amounts of food normally taken by the breast-fed infant (Table I).

TABLE I
AMOUNTS OF MILK TAKEN AT A FEEDING BY AVERAGE-SIZED NORMAL
BREAST-FED INFANTS

AGE	QUANTITY		AGE	QUANTITY	
	OZ.	ML.		OZ.	ML.
1 week	2.5	75	4 months	6	180
2 weeks	3.0	90	6 months	7	210
1 month	4.0	120	8 months	8	240
2 months	4.5	135	1 year	8	240

The chief difference in the intestinal tract of the young infant and the adult lies in the fact that in the infant the ascending and descending portions of the colon are short compared with the transverse colon. The sigmoid at birth is greatly distended with meconium and throughout infancy extends higher into the abdomen than during later life.

Liver

The liver of the full-term infant is large in proportion to the remainder of the body. The lower border is frequently 1 to 2 cm. below the costal margin. It doubles in weight during the first year, thus increasing in size less rapidly than the body as a whole. The gall bladder capacity is smaller in relation to liver size in the newborn infant than in the adult.

The liver of the newborn infant is relatively immature functionally. The rapid blood destruction that occurs during the first ten days after birth would throw an excessive load on even a maturely functioning liver, and some degree of icterus is considered physiologic. More pronounced icterus often is observed in those infants whose cords have

been clamped late, and who thus have more hemoglobin. The liver of the prematurely born infant is even more immature functionally than that of the baby born at term, and jaundice is almost invariable.

Urinary Tract

At the time of birth the kidney is functioning, but not as efficiently as that of the adult. Both the rate of glomerular filtration and the rate of reabsorption through the tubules are lower than in adults. The urinary output is small for the first few days, for the reason that the fluid intake is small (sometimes less than 30 c.c.) and the water loss through the lungs is large. The output of urine during the first 24 hours may be only 5 c.c. and rarely is more than 75 c.c. As the young infant is unable to excrete concentrated urine, nitrogenous waste accumulates in the blood. By the end of the first week the amount of urine is adequate to remove waste. The excretion of uric acid is high during the first week, relatively higher than at any time later. The amount excreted often exceeds that which is easily soluble, and brownish crystals of precipitated uric acid are found in the urine. The maximum uric acid excretion is observed at about the third or fourth day; thereafter the amount decreases rapidly. During the remainder of infancy the daily volume of urine varies from 200 to 900 c.c., depending on the size of the infant, the temperature and humidity of the environment, the fluid intake, and other factors. The urine is often neutral or alkaline, as the infant's normal diet contains an excess of base.

Skeleton (Bone)

Bone in the human being is formed largely from cartilaginous and to a lesser extent from membranous structures. Mineralization starts at many separate points, called centers of ossification, and gradually spreads throughout the structure. Over 800 centers of ossification are formed

in the body, more than half of them appearing after birth. The regularity of appearance of these centers and their rate of mineralization can be used as an index of development. In infancy the rate of ossification of the carpal bones is most used for determination of the "skeletal age." Illness and poor nutrition delay the appearance of these centers. The epiphyses of the long bones have independent ossification centers and grow independently of the diaphyses. Growth of the long bones occurs chiefly at the end of the shaft, between it and the un-united epiphysis.

Chemically, bone is 75 per cent water at birth; the remaining 25 per cent is made up of the organic matrix, mineral salts (a calcium-phosphate-carbonate complex like the apatite minerals), and fat, which is chiefly in the marrow cavities. During growth the mineral content tends to increase and the water content to decrease. The rate at which this exchange proceeds depends on the mineral and vitamin D intake of the baby. The young breast-fed baby cannot maintain the per cent of mineral present in bone at birth. The percentage mineral content, therefore, decreases until about the third month, when it begins to increase and the water content to decrease. The infant who is given cow's milk and vitamin D is able to maintain the percentage mineral content of his skeleton at the birth level up to three months of age, then to increase it fairly rapidly. By one year the water content of bone has decreased to about 60 per cent and the mineral content has increased proportionately. The total skeleton comprises approximately 10 per cent of the body weight at birth. Subsequently the proportion increases to 16 to 20 per cent, at which level it remains throughout life. Growth in length is due entirely to growth of the skeleton; bone growth, in turn, depends on the nutritional status and on heredity. A poorly fed infant may continue to grow in length for a time, but when growth ceases, often a fairly long period of adequate nutrition is required before growth is again resumed.

Skin, Hair, and Subcutaneous Tissues

At birth the skin of the Caucasian infant is bluish red in color, fading to a light pink within two weeks. A yellowish coloration, which may be more or less marked, often develops during the second or third day when destruction of excess of blood pigment begins. Persistent or increasing jaundice after the second week of life is abnormal. During the first month some desquamation of the superficial layers of the skin normally occurs. The sweat glands are relatively inactive at birth, but usually attain full activity within a few weeks. The very young baby also does not develop "gooseflesh," and this means of determining excess coolness of the body cannot be used during the first months of life.

The amount of hair varies greatly among newborn infants. The hair usually is dark, soft, and fine, and may be two inches long. Some babies lose this first hair early and completely, while others seem to lose it only as new hair replaces the first growth. Friction of the head against the bedclothes often keeps the newly grown hair rubbed off and accelerates the loss of the original growth. Many newborn babies have fine downy hair (lanugo) thickly distributed over the body, especially on the back and legs; these fine hairs disappear within a week or two.

The amount of subcutaneous fat differs greatly among newborn infants, but the well-fed infant soon develops a sufficient layer of fat to give to the skin its characteristic soft, springy character. The well baby of two months or more should have a skin pink in color, and moist and elastic to the touch. One of the first evidences of poor nutrition is loss in degree of turgor of the skin; the skin and subcutaneous tissues become loose, flabby, and inelastic.

Lymphatic System

The lymphatic organs are well developed at birth and grow rapidly during infancy. The rate of growth, particularly of the lymph nodes, seems to be rather in response

to infection than related to bodily development. The lymphatic response to infection is characteristic of infancy and early childhood; because of the lack of immunologic defense, infections are likely to reach the lymph nodes.

The thymus is a lymphatic organ which, like the tonsils and adenoids, increases rapidly in size in early life and undergoes regressive changes in late childhood. The thymus atrophies quickly in illness, even of short duration; therefore a thymus of normal size is seen at autopsy only in cases of sudden death. In the past this difference in the size of the thymus in sudden death and after illness gave rise to the concept of an abnormally large thymus as the cause of death, the so-called "status thymo-lymphaticus." This concept is now viewed with considerable skepticism.

The Nervous System

The brain of a newborn infant is large in comparison with the body as a whole. Also it grows more rapidly than the body for the first few months, but growth of the brain begins to slow in midinfancy and is practically complete by seven years of age. The functional development of the brain is far behind its anatomical development and is slowest of any organ, leading to a long period of dependence. Because of the slow development of mental and other brain functions, environment exerts a greater influence on the human than on other species.

Certain motor pathways in the spinal cord do not have their myelin sheaths completed at birth. Until these sheaths develop, those nerve fibers do not function. On the other hand the inhibitory effect of the mature brain on many motor responses to sensory stimuli is much diminished in the immature brain of the newborn. This lack of inhibitory influence accounts for the greater incidence of convulsions in mild illnesses and, in cases of birth injury, for the resulting spasticity. The diminished motor responses due to nonmyelinization tend to neutralize the exaggerated motor responses due to noninhibitory impulses.

As the rate of development of these functions varies considerably, the variation in reflex response among infants of the same age is far wider than among older children.

Tendon Reflexes.—The patellar reflex, or knee-jerk is sometimes exaggerated in early infancy. The jaw reflex is also present at birth. The Achilles reflex often is difficult to obtain in many young infants, but it is present in the majority of older infants.

Superficial Reflexes.—The corneal and pupillary reflexes are present at birth, as are the swallowing and lip reflexes; the last-named gradually disappears. The genital reflex is usually obtainable under one month of age. The abdominal reflex appears irregularly during the first half year of life but is regularly obtained by the end of the first year. The plantar or Babinski reflex produces extension of the toe (dorsal flexion) in most babies until the time of walking (twelve to fifteen months) after which the customary plantar flexion response is obtained.

Although at birth the infant's cerebrum has little influence on his reactions, certain coordinated acts can be carried out even on the first day of extrauterine life. The normal newborn infant is able to suckle and to swallow. He can cry, sneeze, yawn, and hiccup. If his hands are placed around a rod, they will grasp the rod so tightly that when the rod is raised, the baby hangs by his hands. This so-called grasping reflex disappears by three months of age.

The functional development of the nervous system of an infant is of greater importance than the anatomical growth. Increase in functional capacity is shown by a well-ordered progress of increasing cerebral control over body motion. Control of the trunk is learned before control of the extremities; control of the large muscles before that of the small. Early attempts at any muscular achievement are very unsteady; precision of movement is attained very slowly.

The Special Senses

Touch.—The sense of touch is probably the most highly developed of the special senses at birth. A light touch awakens a sleeping young infant immediately, startles the infant already awake. Touching the lips starts the reflex of sucking in the newborn. The lips and mouth are very sensitive tactile areas; much of the infant's investigation of objects is accomplished by putting them in his mouth.

Pain.—Sensitivity to pain is less acute in the young infant than in the older child. The infant often appears wholly indifferent to the reddened and inflamed areas around the site of vaccination or immunization.

Smell and Taste.—These senses are not acute in the young infant. Because of this it appears wiser to start the use of highly flavored fruits and vegetables, as well as of fish-liver oil, before the sense of taste is too well developed. Too frequently, only bland foods are fed as supplements to the infant's diet, and as taste perception develops, more highly flavored foods are rejected.

Hearing.—At birth, the middle ear contains no air and embryonic epithelial tissue persists around the eardrum. Young babies often seem deaf. Soon sharp sounds startle the baby. By the second or third month the infant has learned his mother's voice and may associate certain other familiar sounds pertaining to his comfort, such as splashing of his bath water.

Sight.—The sense of sight is immature at birth. Light during the first few weeks apparently causes discomfort. The muscular control of the eyes is poor so that true binocular vision occurs only by chance in the newborn. The eyes move but not together, nor do they tend to focus on any given object. After about a month, the baby will focus for a few seconds on a bright object held a few inches from his face. His eyes may even follow the object, but for a very short space only. The ability to focus on and follow a slowly moving object dependably is not attained until

between the third and fourth months of age. The eyes occasionally move independently for some time after that. By about four months the baby obviously recognizes familiar objects and persons.

Body Temperature

The newborn infant has not yet developed the ability to maintain the body temperature at the normal level. Even the older infant cannot maintain body temperature during prolonged exposure to extremes of temperature. The maintenance of correct body temperature in an infant must be the responsibility of the attending adult. Covers should be light but warm, and should not impede motion. During periods of excessive heat, tepid baths and sprinkling the baby's bed with warm water are helpful in keeping body temperature from rising above the normal level. The usual tendency of attendants appears to be in the direction of keeping the baby too warm.

Skeletal Muscle and Motor Behavior

Skeletal muscle comprises approximately 25 per cent of the body weight of the infant in contrast with 40 to 45 per cent of the body weight of the adult. This low proportion of total muscle weight to total body weight persists throughout the period of infancy.

At the time of birth, and during the first week or two, the muscular movements of the infant are not very active or well coordinated. He will cry, draw up the legs, and grasp any object placed in the palm of the hands. The one set of muscular movements in which even the newly born normal infant shows proficiency is sucking. When placed at the breast or when a nipple is put in the mouth, the infant sucks vigorously and effectively.

Motor behavior throughout infancy depends in large measure on mental development and the state of health.

When these factors are normal, the motor development of the infant progresses regularly, the rate of progress being slow for the first months and progressively more rapid subsequently. The exact age of achievement of certain abilities depends in considerable measure on the venturesomeness of the individual infant and on the amount of attention given. In general, the following progression of motor development may be expected.

For the first three or four weeks an infant is unable to change his position or raise his head when on his back. Random movements of arms and legs occur and may be vigorous in a strong baby. By the third month an infant can hold the head erect when the body is supported. Inability to do so by the end of the fourth month is usually indicative of physical or mental abnormality. At two to three months the muscles of the eyes coordinate fairly well, and by three to four months the eyes will follow a bright, moving object held a short distance from the face. When this occurs, the baby begins to reach awkwardly for objects. When objects are placed in the hand, his grasp is still inaccurate, but at five to seven months the baby attains fair accuracy in reaching for and holding objects.

The neuromuscular maturity of the mouth and throat is sufficiently developed by the fourth month so that the infant can be taught to take semisolid food from a spoon. Some infants can learn this procedure earlier, but if spoon feeding is begun too early, so that the infant is too immature to master it, undesirable behavior reactions occur which may make spoon feeding difficult for months.

By the fifth or sixth month a baby can sit alone for short periods, and sit well with support. At seven to eight months he can sit unsupported and maintain a straight back. Many babies, particularly those often played with by adults, will attempt to stand as early as five months,

when supported. Few babies stand well, even with support, before the seventh or eighth month, but most infants do so easily by nine or ten months of age. By the seventh or eighth month most babies will creep a short distance, though some babies never creep. By nine months most babies will pull themselves to standing in the crib or beside furniture. Walking with support begins at about this period, but walking alone is not achieved until twelve to sixteen months of age and is accompanied by frequent falls.

Blood

The relative blood volume, the red cell count and the hemoglobin concentration are higher at birth than at any time later. The blood constitutes about 12 per cent of the total body weight in the newborn infant, and at eight to ten months of age it is still 10 per cent or more of the body weight; in the adult, blood accounts for about 9 per cent of the weight of the body.

In the case of infants who have been underfed or who have become dehydrated, the absolute volume of the blood is decreased, but not necessarily to the same extent as the decrease in weight. Decrease in the blood volume regularly results in marked impairment of the circulation. We have observed malnourished and dehydrated infants in whom the volume flow of the blood in the extremities was less than one-tenth of the normal. The volume flow in the internal organs, although not susceptible to accurate measurement, appears also to be decreased, and results in a diminished degree of absorption from the gastrointestinal tract, as well as functional incapacity of the heart and some of the secreting organs.

The hemoglobin content of the blood at birth varies from 15 to 25 grams, averaging about 22 grams to each 100 milliliters. It seems generally agreed that the high hemoglobin content is a result of the low oxygen tension in the

placenta. After the infant's lungs begin to function in air, this quantity of hemoglobin is unnecessary, and the excess is destroyed. The period of "physiological blood destruction" lasts for eight to ten weeks. The destruction is most rapid during the first week or two, and the rapid conversion of blood pigment to bile pigment is probably responsible, at least in part, for the icterus of the young infant.

Much of the iron of the destroyed hemoglobin is conserved by the infant and used later. The minimum hemoglobin level is reached between eight and twelve weeks of age. By this time the hemoglobin has decreased to a level of from 10 to 12 grams to each 100 milliliters of blood. A slight rise then usually occurs, perhaps under the stimulus of the increased store of iron in the liver. After about the fifth or sixth month, unless some form of iron is fed, the hemoglobin decreases steadily as the body stores become exhausted. The iron saved from the destroyed hemoglobin amounts to from 100 mg. to a maximum of 300 mg. with an average of about 150 mg. Approximately another 50 mg. is already stored in the liver at birth. By six months of age both of these sources are used up in maintaining hemoglobin. Milk, either human or cow's, contains little iron; other sources must be added by six months, or soon thereafter, to prevent development of nutritional anemia.

Prematurely born infants have small iron reserves because of their smaller total blood volume and because storage of iron in the fetal liver does not occur until toward the end of gestation. These infants are thus particularly liable to become anemic, and additional sources of iron should be given early in life.

The red blood corpuscles of the newly born full-term infant usually number well over 5,000,000 for each cubic millimeter and are occasionally as high as 8,000,000. Nucleated red blood cells may be present during the first

week of life, but they are not found under normal conditions after this time. The number of red blood corpuscles diminishes rapidly during the first few weeks to a level of 3,500,000 to 4,500,000 for each cubic millimeter. During the latter half of infancy the red blood cell count varies usually between four and five million for each cubic millimeter, and the hemoglobin from 11 to 14 grams for each 100 milliliters. One frequently observes pale, poorly nourished infants who have normal red blood cell counts. In the condition of anhydremia the red blood cell count usually is distinctly above normal, although the infants are actually suffering from greatly decreased total blood quantity. It is essential that one should distinguish between anemia and oligemia. An infant with a normal red cell count or a normal proportion of hemoglobin may still have a greatly decreased blood volume and be in need of transfusion.

The white blood cell count is variable in early infancy and fluctuates widely. At birth the white corpuscles vary from 15,000 to 25,000 for each cubic millimeter, with polymorphonuclear cells predominating and with many immature forms. Soon the number decreases to between 5,000 and 20,000, with lymphocytes predominating; the average is approximately 12,000 for the first two years, with large fluctuations. Prematurely born infants at birth show a large number of immature cells of all types, which may persist in the circulation for several months.

The percentage of polymorphonuclear cells during the first few days of life varies from 70 to 75 per cent of the total white cell count. By the end of the second week this percentage decreases to 30 or 40, and at the same time the percentage of lymphocytes increases to from 50 to 65 per cent. It is not until the fourth or fifth year of life that the percentages of polymorphonuclear neutrophils and mononuclear cells become equal. Thereafter a gradual increase occurs in the polymorphonuclear cells until the adult proportion of 60 to 75 per cent is attained about the twelfth

year. The percentages of eosinophiles and basophiles in the blood of the infant do not differ materially from those in adult blood. Differential white blood cell counts by the Schilling technique show distinct variations from the adult hemogram; the percentage of lymphocytes is higher, there are more stab cells and juvenile forms and a lower percentage of segmented forms; in the presence of infections in infants, a greater "shift to the left" may be observed. The following table shows for comparison the Schilling hemograms during infancy and during adult life.

TABLE II

	BASO- PHILES	EOSINO- PHILES	LYMPHO- BLASTS	MYELO- BLASTS	MYELO- CYTES	JUVE- NILES	STAB	SEG- MENTED	LYMPHO- CYTES	MONO- NUCLEAR
Infant	0.1	1.3	0	0	0	2-5	4-7	20-40	50-75	4-9
Adult	0.1	2.4	0	0	0	0	3-5	55-65	20-25	4-8

The blood may show distinct differentiation into the usual blood types and groups from the time of birth. It is therefore as necessary to match blood for transfusion of the infant as in the case of older persons. The mother's blood is not always compatible with that of the infant.

Endocrine Glands

In the normal infant, both the thyroid and the islands of Langerhans begin activity before birth. Congenital absence of the thyroid gives rise to cretinism; hypoplasia, to infantile myxedema. Infants of diabetic mothers may have increased activity of the islands of Langerhans, presumably compensating for the maternal deficiency; in such instances hypoglycemia must be combated until readjustment of insulin secretion occurs.

Occasionally tetany is encountered in the neonatal period. The cause presumably is suppression of the infant's parathyroid secretion brought about by maternal

oversecretion. In all such cases the function of the infant's parathyroid is later re-established, but in the meantime the convulsions require specific or symptomatic treatment.

The hormones that cause lactation in the mother pass into the fetal circulation and in many instances cause breast secretion in the infant. Lactation in the infant is transitory and does not pass beyond the colostrum stage. Both sexes are affected.

The thymus has been considered as having an endocrine function. The thymus is a lymphatic organ and the evidence of endocrine activity is unconvincing to many observers.

CHAPTER II

ENERGY METABOLISM

The metabolic processes of the infant are of essentially the same character as those of the adult, but are relatively much more active. A large intake of food for each unit of body weight is necessary in the case of infants, not only to supply material for growth, but also to allow for a very active energy exchange.

The living body, like a machine, requires fuel and materials for repairs and replacements. The growing individual has an additional requirement for growth. These needs are met by food, which must have a sufficient fuel value, that is, must supply enough calories. It must also provide certain minimum amounts of protein or the constituent essential amino acids, carbohydrate, mineral salts, water, vitamins, and possibly certain fatty acids in the form of their neutral glycerides.

The Significance of Calories

No matter what the type of food, it must provide sufficient energy or fuel value to supply body heat and the energy necessary for the activities of the heart, lungs, skeletal muscles and digestive organs. When food is utilized in the body as fuel, the energy derived from it is ultimately given off in the form of heat. For this reason the amount of heat liberated by the body is a measure of the amount of food "burned" in the metabolic processes. This value may most conveniently be expressed in terms of heat units or *calories*.

The calorie is a measure of energy expressed in terms of heat. To illustrate, let us take a simple food, such as sugar. If sugar is burned, heat is liberated; at the same time oxygen is taken up and carbon dioxide and water are produced. The exact amount of heat given off by this

combustion and the amounts of oxygen consumed and carbon dioxide produced may be measured in a calorimeter. In such an apparatus a known amount of sugar, say one gram, is placed in a small, water-tight, metal "bomb," and oxygen under pressure is introduced so as to insure complete combustion. The bomb is immersed in a known amount of water, and the sugar is ignited by an electric spark. The increase in the temperature of the surrounding water after the burning of the sugar is noted and subsequently the oxygen and carbon dioxide contents of the gas in the bomb are analyzed. If one gram of sugar is burned, and the volume of water surrounding the bomb is one liter, it is found that the temperature of the water is raised approximately 4° C. Since a large calorie (the unit used in metabolic work) is defined as the amount of heat necessary to raise the temperature of one liter of water 1° C., the gram of sugar in burning liberates four calories.

If we could imagine a small steam engine placed inside the bomb and the sugar burned under the boiler of this engine, so as to make it run, the heat of the burning sugar would be converted into mechanical energy which would, in turn, be converted back into heat, so that when the whole system once more had come to rest, it would be found that the temperature of the outside water would have been raised to exactly the same extent as when the sugar burned directly, without first producing mechanical energy. A calorie, in terms of mechanical energy, is equivalent to approximately 3000 foot pounds, that is to say, the energy of one calorie is sufficient to raise 3000 pounds or a ton and a half to a height of one foot, or to raise one pound to a height of 3000 feet.

When sugar is "burned" in the body, it produces exactly the same amount of heat as when burned in the air, and exactly the same amounts of oxygen are utilized and carbon dioxide given off. The energy produced may be converted into such mechanical work as muscular contraction, but ultimately appears as heat. If an individual is put into a

large enough calorimeter chamber surrounded by water and if the air entering and leaving the chamber is analyzed and the food and excretions are analyzed, the exact amount of sugar or other foods utilized in the body may be determined, and from these findings the amount of heat which should be produced can be calculated. It has been found that the amount of heat so calculated is the same as that actually produced and measured by the rise in temperature of the surrounding water.

All foods do not have the same fuel value. One gram of fat has a fuel value of a little over 9 calories in the calorimeter, and also in the body. Protein does not form the same end products when burned in the air as in the body because the amino groups of proteins are not oxidized in the body beyond the stage of urea, whereas protein burned in the calorimeter is completely oxidized. The fuel value of protein in the body is about 4 calories for each gram. Individual sugars, fats and proteins have slightly different fuel values. The figures given above are rough averages.

During life, the human body continuously gives off heat, the source of which is the combustion of proteins, carbohydrates, and fats, together with small amounts of other foodstuffs such as alcohol and organic acids. Even when no food is taken, the body continues to liberate heat, which comes from the utilization of glycogen of the liver and muscles, fat of the subcutaneous tissues and other fat depots, and to a lesser extent of protein of the muscles and other organs.

The amount of fuel consumed, as measured by the oxygen intake and carbon dioxide output, or by actual heat production, bears a definite relationship to the age, weight, and size of the body.

Basal Metabolism

The term "basal metabolism" is used to designate the heat output of an individual at complete rest and in the postabsorptive state as regards food. The heat produc-

tion that occurs under these circumstances represents the combustion of food materials by the "active protoplasmic tissues" of the body when they are as nearly at rest as is possible during life in the normal person. A major component of these active tissues is muscle. Consequently, when the muscle mass is in its normal proportion to the remaining active tissues, the basal metabolic rate is proportional to the amount of muscle tissue. Since creatinine excretion is proportional to muscle mass, some observers have calculated the basal rate in relationship to the creatinine output. This procedure apparently is not applicable when marked muscle wasting exists from any cause. For everyday practical use it is more convenient to calculate basal rates in terms of factors more easily measured than is the excretion of creatinine. In the growing infant and child, the amount of active tissue is proportional to the age, weight, height and surface area when the child is of normal or average size for his age. For persons beyond infancy, it is generally customary to determine the basal rate in terms of surface area as calculated from the height and weight. For the infant, it is customary to consider the basal rate in terms of weight alone, even though the rate in terms of body length is probably more accurate.

The basal metabolism of normal infants during the first year of life has been found to average 55 calories a day for each kilogram of body weight (25 calories for each pound). That is to say, a normal infant weighing 5 kilograms (or 11 pounds) would be expected to liberate approximately 275 calories in 24 hours. Unless the food taken in has a caloric value at least this great, the body itself is consumed as fuel. If food having a caloric value just equal to the basal metabolism were given, it would be found insufficient because the utilization of food in itself leads to a certain increase in the heat output. Carbohydrate utilization raises the rate of metabolism very little, fat somewhat more, and protein very considerably. This effect of food in increasing the rate of metabolism and heat output is

termed the "specific dynamic action." The average increase in heat production due to the taking of a mixed diet is considerably less, however, than the sum of the increase due to each component. It has been considered that the specific dynamic action of cow's milk is greater than that of human milk because of its higher protein content, but probably the specific dynamic action of each is less than 10 per cent of the basal metabolic requirement.

Allowance for Activity

The preceding calculations are based on the assumption that the infant remains at complete rest; but no infant remains at complete rest throughout the twenty-four hours, and any activity necessarily calls for an increased expenditure of energy and a correspondingly greater heat output. Vigorous crying, while it lasts, may increase the heat output by over 100 per cent. On the basis of complete metabolic experiments carried out with infants over long periods of time, it has been determined that a fair extra allowance for activity in the case of average infants is from 20 to 25 calories a day for each kilogram of body weight (10 calories for each pound). Very active infants may use up more than 80 calories for each kilogram by extra activity, and very placid ones as little as 10 or 15.

Allowance for Growth

These figures make no allowance for growth. The amounts of food required for the building up of new tissue cannot be estimated by the heat output since the food used for this purpose is not burned as fuel but is stored. An estimate may, however, be made on the basis of the known average daily increase in weights of infants and the proportions of this increase which are due to protein and fat deposition. These estimates have been checked by observations on the actual amounts of food needed to maintain stationary weight and the additional amounts necessary to bring about a normal gain. It has been found that an al-

lowance of 15 to 20 calories a day for each kilogram of body weight (8 calories for each pound) is adequate for an infant gaining at an average rate. Infants who are undernourished as the result of long-continued inadequate feeding may gain very rapidly in weight and deposit two or three times as much protein and fat as infants in average nutritional condition. These undernourished infants, therefore, require a growth allowance of food much in excess of the average.

Allowance for Unutilized Food

Food taken in by mouth is never completely digested and absorbed, but a certain amount is lost by way of the bowel. The quantity so lost under normal conditions is equivalent to 10 or 15 per cent of the intake, which in the case of the average infant corresponds to 10 or 15 calories daily for each kilogram of body weight (5 to 7 calories for each pound). In the presence of diarrhea or digestive disturbance, the loss of food by way of the bowel may be as great as 30 or 40 per cent of the intake.

The Total Energy Requirements

To sum up, the total daily caloric requirements of the average normal infant during the first year of life are as shown in Table III.

TABLE III
ENERGY REQUIREMENTS IN INFANCY

Basal resting metabolism	55 calories per kilogram (25 per pound)
Allowance for specific dynamic action	10 calories per kilogram (4 per pound)
Allowance for activity	25 calories per kilogram (10 per pound)
Allowance for growth	15 calories per kilogram (7 per pound)
Allowance for unutilized food	10 calories per kilogram (6 per pound)
Total	115 calories per kilogram (52 per pound)

Observations on normal infants have confirmed the preceding calculations. The actual intake of food by young, healthy breast-fed or artificially fed infants, gaining in weight at normal rates, has been measured and the caloric value has been found to average from 110 to 115 calories a day for each kilogram, or 50 to 52 calories for each pound.

The preceding figures are averages for the entire first year. Actually there is considerable variation, even in the case of the same infant at different periods during the first year. For the first three or four weeks after birth the metabolism is relatively low; but by the end of the first month the food requirement is as much as 120 or 130 calories for each kilogram (55 to 60 to the pound). Thereafter the requirement slowly diminishes to about 110 calories for each kilogram (50 calories to the pound) by

METABOLISM DURING FIRST YEAR OF LIFE

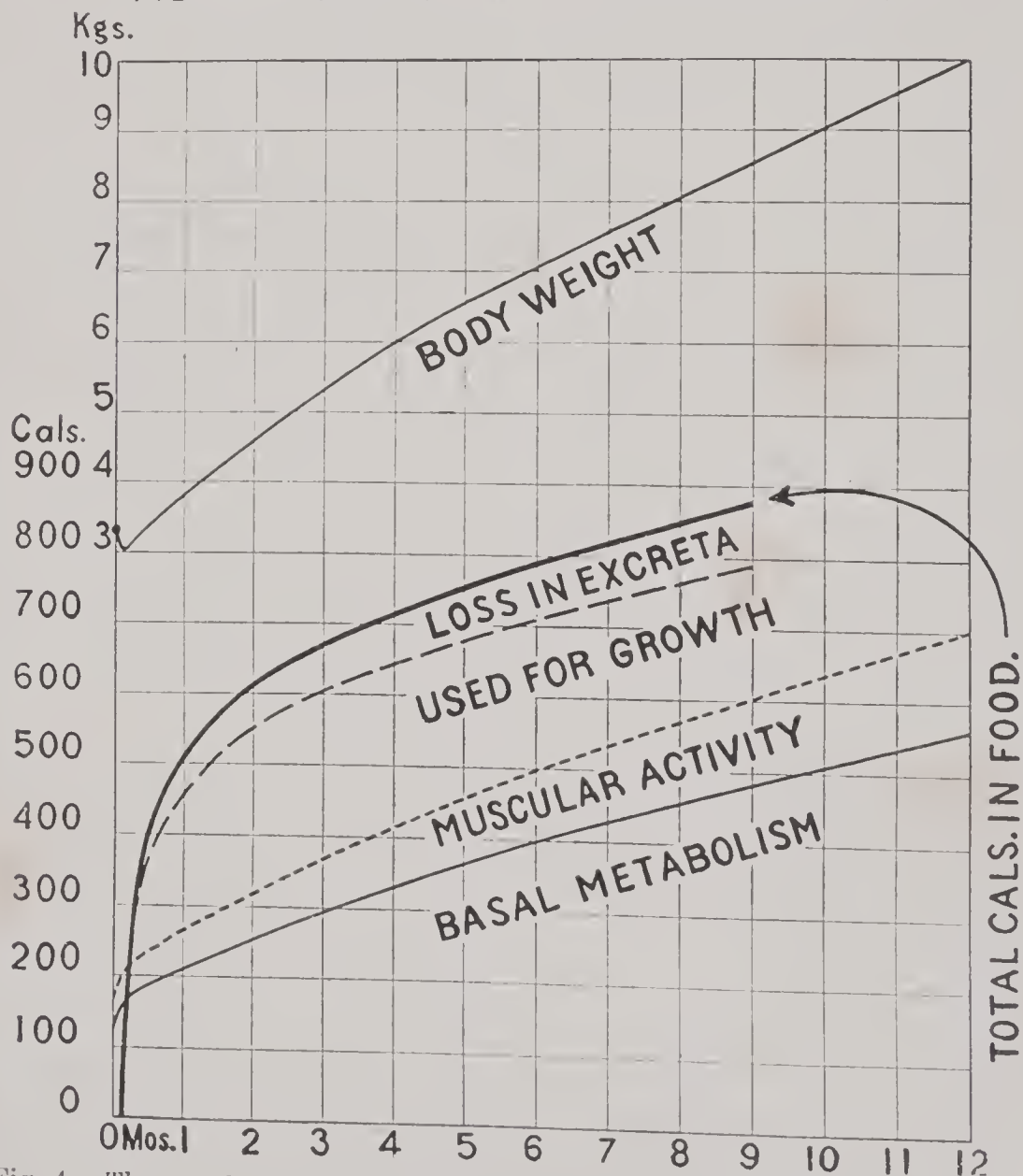


Fig. 4.—The metabolic requirements of infants. (Talbot, F. B.: *Am. J. Dis. Child.* xviii, 229, 1919.)

the sixth month, and 90 to 100 calories for each kilogram (40 to 45 to the pound) by the end of the first year.

The total energy requirements of the average infant are expressed graphically in Fig. 4 (Talbot).

The figures given must not be taken as literally applying to every infant. They serve merely to indicate the approximate total food requirements. Strong, active infants have considerably greater energy requirements than weaker, apathetic ones of the same age and weight. The total food requirements during cold weather are greater than during warm weather. Infants who are markedly underweight or small for the age have greater food requirements than normal infants.

For underweight infants the *total* food requirement may be as great as that of normal infants who are of the same age but weigh much more. For example, a normal infant six months of age, weighing 15 pounds, would require approximately 800 calories a day. A malnourished infant, 6 months of age, weighing only 7½ pounds, would also require very close to 800 calories a day, which would be the equivalent of 100 calories for each pound, instead of 55 as in the case of the normal infant. The reason for this difference is that the thin infant and the well-nourished one might have approximately the same amount of active muscular tissue and the same-sized heart and lungs. The chief difference would be in the layer of adipose tissue, a relatively inert substance, requiring but little food for its maintenance, the fat acting very much as an overcoat and preventing loss of heat. It is the active protoplasmic tissue of the body which determines the food requirement, rather than the actual weight. Accordingly, in attempting to arrive at an estimate as to the proper food requirement of an undernourished infant, the first step is to calculate what would be the approximate weight were the infant normal. In order to do this, reference should be made to the normal weight curves, taking into consideration the birth weight.

In the case of excessively fat infants, the reverse condition holds true. The fat infant does not require much if any more food than an average-weight infant of the same age. His caloric requirement for each unit of body weight, therefore, may be less than that of the average infant.

In the case of an individual infant, it is impossible to state exactly how many calories will be needed in order to bring about a normal gain in weight. One can, however, obtain a general idea on the basis of the principles just laid down. It is important that the caloric value of the food should be calculated in every instance, in order to avoid gross errors of underfeeding. The average total daily caloric need of normal infants at various ages is approximately as follows:

1 month	500 calories	9 months	900 calories
2 months	600 calories	One year	1000 calories
4 months	700 calories	Two years	1200 calories
6 months	800 calories		

The final test of the adequacy of the diet is the response on the part of the infant. It is, at times, necessary to give food of a considerably higher caloric value than would be anticipated. *The giving of food of a caloric value too low to meet the infant's needs is by all odds the chief cause of failure in infant feeding.* Unless the energy requirements are met, the infant will fail to do well, even though the food may be easily digestible and otherwise admirable in composition.

It is essential that food having a sufficient caloric value be given, but the form in which the calories are supplied is also of importance. If an infant were to receive the total caloric requirement in the form of pure carbohydrate, the maintenance of life and health over a prolonged period would not be possible. Certain food constituents are essential to supply the wear and tear of body tissues, to build up new tissues, and to assure the proper utilization of the energy provided. The diet must be correct in quality as well as quantity. In the succeeding chapters the significance of the individual food constituents is considered.



CHAPTER III

PROTEIN METABOLISM

Protein is essential for maintenance of life and for building body tissue. The protein need of the infant is proportionately greater than that of the adult because of rapid growth.

The Composition of Protein

All proteins do not have the same composition or the same value in nutrition, although they are all composed of combinations of amino acids united in complex chains. There are at least twenty-two amino acids which exist in varying proportions in the proteins. Only ten of these have been shown to be essential for the experimental animal. Human adults have been maintained in nitrogen equilibrium with only eight of the ten necessary for animals. The number necessary for the human during the period of growth or for maintenance of health of the adult has not been determined, though the need probably is much the same as for the animal. Cystine is an amino acid that is not essential, but it is useful in that to some extent it can replace methionine, which is an essential amino acid. Both these substances contain sulfur. Certain essential amino acids are entirely lacking in such proteins as gelatin and in many of the vegetable proteins. Inasmuch as the human body is incapable of synthesizing a number of the amino acids, incomplete proteins or those deficient in these essential amino acids cannot be converted into body protein unless the lacking amino acids are supplied from some other source. For example, if a protein such as gelatin, which is deficient in tryptophane and sulfur-containing amino acids, is fed as the sole source of protein, no new body protein at all can be built up, even though the other amino

acids are present in adequate amounts. Proteins containing small amounts of essential amino acids can build up only proportionately small amounts of body protein. The amount of protein needed by the individual, therefore, depends on the character of the protein given. Proteins that have a composition closely simulating that of the body may be converted almost quantitatively into body protein, and therefore have a greater value in nutrition than those proteins of which the composition differs widely from that of body tissues.

The young infant customarily receives his protein from either human or cow's milk. The proteins of milk may be divided into two main groups, whey protein or lactalbumin and curd protein or casein. Lactalbumin differs materially from casein in its composition; it has been considered the more valuable of the two proteins. Human milk contains less protein than cow's milk, but of the total protein present almost 60 per cent is lactalbumin, as compared with about 15 per cent in cow's milk. Though the proteins of both these milks are of high nutritional value, that of cow's milk has long been considered as of less biologic value than that of human milk. For this reason it has been believed necessary to feed a larger amount of cow's milk protein than of human milk protein. More recent evidence casts some doubt on this view. Nevertheless, babies who receive cow's milk formulas with percentage composition similar to that of human milk do not grow so well as those receiving human milk or cow's milk formulas of customary composition. Possibly the differences in growth are dependent on factors other than protein.

The Protein Requirement of Infants

The baby who is thriving with breast feeding receives from 2 to 2½ grams of protein daily for each kilogram of body weight. Consequently this amount of protein is generally considered as optimum when supplied by human milk. With this requirement as a basis, and with continued

acceptance for the present of the concept that cow's milk protein may be as much as 20 per cent inferior to human milk protein. $2\frac{1}{2}$ to 3 grams of cow's milk protein for each kilogram of body weight would be required to supply an optimum amount. As a matter of fact, customary cow's milk formulas for young babies supply $3\frac{1}{2}$ to $4\frac{1}{2}$ grams, or more, for each kilogram of body weight. Stated in another way, the protein of human milk supplies approximately 9 per cent of the total calories, while the protein of cow's milk formulas supplies approximately 16 per cent.

It is difficult to find a satisfactory basis for determining the optimum protein requirement of the infant. When babies are fed the larger quantity of protein of the usual artificial feeding, nitrogen retention is greater, resulting in a greater increase in active body tissue, since nitrogen is not stored except in such form. In the case of babies who are artificially fed in the customary manner, the percentage nitrogen content of the body, when plotted, increases in a curve smoothly continuous with the curve of increase during fetal life; for babies who are breast fed, the curve of increase during fetal life changes course sharply at the time of birth to the extent that little or no percentage increase in nitrogen takes place for a period of months. Subsequently, with the addition of other foods and with weaning, the curve of percentage increase rises to meet the level maintained by the artificially fed baby. Trying to determine which of these two types of curve is the more desirable leads only into a field of speculation.

Accepting $3\frac{1}{2}$ to $4\frac{1}{2}$ grams of protein for each kilogram of body weight as a suitable daily intake for the artificially fed baby, it is found that this amount is obtained by the infant when he receives daily a quantity of cow's milk equivalent to from one-tenth to one-eighth of his body weight, or from $1\frac{1}{2}$ to 2 ounces for each pound. In the case of undernourished infants, the normal rather than the actual body weight should be taken as the basis of the calculation.

In young infants, a very large proportion of the protein taken in with the food is converted into body tissues and retained, hence does not appear as nitrogenous end products in the urine. In young breast-fed infants, receiving relatively small amounts of protein, as much as 80 per cent may be stored in this way. The growing infant is normally in a state of positive nitrogen balance, whereas the fully grown individual excretes in the urine as much nitrogen in the form of end products as is contained in the ingested protein.

Protein Under- and Overnutrition

When the protein content of the diet is insufficient, growth is slow, resistance to infection is decreased, the musculature becomes flabby, and secondary anemia develops. As a result of prolonged protein underfeeding, the concentration of blood plasma protein is decreased; this is associated with the development of edema (nutritional edema), which may be of an extreme degree.

A moderate excess of protein in the diet above the amounts mentioned apparently does no harm. It is well digested and absorbed and causes no general disturbances. When a very great excess of protein is fed, especially when only small amounts of water are given, anhydremia or dehydration of the body may occur and may be associated with the development of fever and toxic symptoms. Such an effect is not likely to be observed unless the protein given is in considerably higher concentration than in undiluted cow's milk. The feeding of whole cow's milk enriched with additional protein, or the feeding of highly concentrated milk, such as undiluted evaporated milk or concentrated dried milk suspensions, may result in dehydration unless additional fluids are given either by mouth between feedings or parenterally. Under ordinary conditions, however, there is little likelihood of an excess of protein in the diet causing harm.

One effect of high protein in the diet is to lead to an increased secretion of alkaline intestinal juices. At the same time, little if any acid is produced by bacterial decomposition of protein, so that the stools become alkaline in reaction and constipated in type. Casein is more effective than lactalbumin in bringing about this phenomenon, as it appears to be less easily attacked by intestinal bacteria, and furthermore leaves an alkaline residue with a high calcium content. This effect is especially desirable in cases of fermentative diarrhea.

Proteins Other Than Those of Milk

During the first few months of life the infant's protein need is covered almost entirely by milk proteins. Later, the additional foods given (egg yolk, vegetables, fruits, cereals, and meat products) supplement the protein intake to some extent, but milk still remains the chief source.

The protein of the soybean, although not equal in nutritional value to milk protein, is nevertheless a fairly suitable protein for the feeding of the infant and may be substituted for the proteins of milk provided a somewhat larger amount is given. Soybean preparations find their special field of usefulness in those cases in which the infant is allergic to cow's milk proteins. Satisfactory nutrition may be maintained through the use of soybean preparations.

Gelatin is an incomplete protein. It has occasionally been used in infant feeding for the purpose of providing additional calories, and also because when added to milk in sufficient amounts it modifies the character of the curds, rendering them somewhat smaller and softer. For this latter purpose rather large amounts of gelatin are required. The curds may be modified more effectively by other methods. The feeding of gelatin decreases peptic digestion and sometimes results in the passage of foul stools. There would, in general, appear to be no very good reason for gelatin additions to the infant's dietary.

Protein, under normal conditions, is completely broken down into amino acids before absorption from the intestinal tract, but in the case of young infants, and especially of those suffering from severe gastrointestinal and nutritional disturbances, some incompletely digested protein remnants may be absorbed directly into the blood stream and the amount absorbed may be sufficient to sensitize the infant against the particular protein, so that subsequent feedings of the same protein may result in allergic phenomena. The subject of allergy is considered in more detail in Chapter XXVI.

CHAPTER IV

CARBOHYDRATE METABOLISM

Carbohydrate is an essential constituent of the body and is necessary to life, though theoretically it is not an essential constituent of the diet. Carbohydrate becomes available to the body from the breakdown of protein and, to some extent, of fat; these sources ordinarily contribute minor amounts of the total carbohydrate intake. Normally most of the energy used by the body comes from oxidation of carbohydrate and fat. The body always oxidizes carbohydrate in preference to fat. Also, the capacity of the infant to utilize fat is lower than that of the adult and his energy requirement is high. It is thus especially desirable for the infant that carbohydrate provide a generous proportion of the total calories for energy and the amount of carbohydrate customarily fed to a baby is considerably in excess of any calculated requirement. The breast-fed baby receives daily about 12 grams of carbohydrate for each kilogram of body weight, an amount which supplies approximately 40 per cent of the caloric value of his food. Artificially fed babies customarily receive an amount of carbohydrate equivalent to approximately one one-hundredth of the body weight, with a general range of 8 to 14 grams for each kilogram. Of this quantity, about 40 per cent of the sugar is natural to the milk and 60 per cent is added sugar. Because of the high energy need of the infant and the relative ease with which much of this need can be supplied by carbohydrate, it is perhaps fortunate that the infant is capable of taking somewhat more carbohydrate for each unit of weight than the adult without development of glycosuria.

Carbohydrates are chemically much simpler substances than proteins. They are composed only of carbon, hydrogen and oxygen, the latter two elements being in approximately the same proportions as in water. The simplest

carbohydrates are the monosaccharides, such as dextrose, levulose and galactose. Two molecules of any of these joined together form the disaccharides, examples of which are sucrose, lactose and maltose. Sucrose, lactose, and mixed carbohydrate preparations containing maltose are the forms of carbohydrate usually fed to infants. In addition to these simple sugars, there are carbohydrates containing a larger number of molecules of the monosaccharides, as, for example, starch, which is composed of a very large number of molecules of dextrose. Intermediate between starch and the disaccharides are the dextrans, which represent partially broken-down starch molecules.

All carbohydrates are ultimately utilized by the body in the form of monosaccharides or simple sugars, usually dextrose. The conversion to monosaccharides occurs in the intestinal tract. Only when the intestinal mucosa is injured does any carbohydrate, except in the form of the monosaccharides, pass into the circulation. Neither sucrose nor lactose can be utilized when injected into the blood stream or absorbed from the intestinal tract. Each of these is excreted unchanged. Maltose, on the other hand, may be partially utilized, and the monosaccharides, dextrose, levulose, and galactose, are completely utilized when introduced into the circulation.

During the absorption of carbohydrate from the intestinal tract, there occurs a moderate rise in the dextrose content of the blood. During fasting the dextrose content of the blood usually has a range of 60 to 80 mg. for each 100 ml. At the height of digestion of a meal containing carbohydrate, the blood-sugar level does not usually increase more than 20 to 50 mg. above the fasting level. With an increase to as high as 150 mg. or more, which rarely occurs in the normal individual except when huge amounts of carbohydrate are given, some dextrose passes out into the urine.*

*The values for blood dextrose are based on the "true" sugar of the blood. Some of the methods of determination used in the past give values slightly higher.

The level of the blood sugar is kept within these narrow limits by an efficient regulatory mechanism. Dextrose which is needed for fuel is promptly burned in the tissues, the remainder is stored in the liver and muscles in the form of glycogen or animal dextrin, or else is converted into fat and stored as such in the subcutaneous tissues and the other fat depots of the body. During starvation the stored glycogen is reconverted into dextrose, which is poured into the blood and utilized as food. In long-continued starvation the glycogen reserves of the body may be depleted so that the level of the blood sugar falls. Some additional dextrose becomes available through the metabolism of protein, approximately 60 per cent of the latter being convertible into dextrose. This tends to prevent the blood sugar from falling very far below the normal level. A fall in blood sugar to 20 to 30 mg. below the fasting level is associated with hunger contractions of the stomach, weakness, irritability and at times trembling and profuse perspiration. With a fall of more than 20 to 30 mg., especially if the fall has been rapid, generalized convulsions followed by coma are likely to occur. Such extremely low blood-sugar levels are not observed in normal infants except after the administration of an overdose of insulin. The severe symptoms are all promptly relieved by the administration of sugar by mouth or by dextrose intravenously.

Infants born of diabetic mothers not infrequently, during the first week of life, show marked symptoms of hypoglycemia and the blood sugar may be abnormally low. This condition is due to an overproduction of insulin on the part of the infant, apparently in compensation for the mother's lack of insulin. Adjustment of insulin production to food intake ultimately occurs, but for a period of time it may be necessary to administer dextrose at short intervals. Some tendency to hypoglycemia is often exhibited by normal infants during the first week of life and before an adequate food intake has been established.

Insulin is essential for carbohydrate utilization. Too little insulin results in high blood sugar values and an excess may cause too rapid disappearance of carbohydrate from the blood. At one time insulin was given occasionally to undernourished infants with the idea of lowering the blood sugar purposely in order to increase the appetite and bring about a better utilization of carbohydrate in the body.

Carbohydrate is the most readily available source of energy for the body and may replace either protein or fat for this purpose. The administration of adequate amounts of carbohydrate spares body protein from destruction during periods of underfeeding. Carbohydrate, however, cannot entirely replace protein in the diet as it cannot build up the essential protoplasmic structures of the body. In the case of infants who are much underweight, it is often desirable to feed large amounts of carbohydrate, which procedure is without danger provided the carbohydrate is not of a readily fermentable type and provided adequate amounts of protein and of other dietary essentials are fed at the same time. The administration of a diet consisting mainly of carbohydrates and deficient in protein and minerals, may result in a rapid increase in weight, which is due largely to water retention. Infants so fed are likely to be pale, flabby and waterlogged, and to lack resistance to infections. The effects of such a diet are due not so much to an excess of carbohydrate as to deficiencies in other constituents. Sweetened condensed milk and malted milk are examples of foods of the type just mentioned.

The carbohydrates commonly used in infant feeding are lactose, sucrose, maltose-dextrin mixtures, dextrose and starch. Any of these forms of carbohydrate ultimately serves the same functions in nutrition. In the choice of a carbohydrate, however, certain characteristics should be taken into consideration which are different for the various sugars. One has to consider the ease of digestion and

absorption, the degree to which the carbohydrate is fermented in the intestinal tract, and the degree to which it irritates the intestine.

Lactose (Milk Sugar)

Lactose is the sugar occurring naturally in the milk of all mammals. For this reason there are some who consider milk sugar as the appropriate carbohydrate for routine addition to milk formulas. Though there may be more or less definite indications for the use of milk sugar at times, the theoretical reason that it is the naturally occurring sugar of milk is vague and unsatisfactory. If there is any special need for lactose by the body (and it has never been proved that there is), such a need is met by the lactose present in the milk which invariably forms the basis of the infant's diet. Experience has shown that the remainder of the carbohydrate may be supplied by some other sugar with no deleterious effects on nutrition. Also, babies have been reared successfully with a lactose-free diet; these babies developed normally and had excellent retention of calcium, phosphorus, and nitrogen.

Milk sugar is not so sweet as sucrose. This property is advantageous in that the infant does not become accustomed to excessive sweetness of the food. Lactose is easily fermented by most of the normal intestinal bacteria of infants with the production of various acids, including lactic acid. Lactose is not digested and absorbed as rapidly as some other sugars, and in consequence a portion may reach the large intestine before absorption. In this location it is quickly broken up by the intestinal organisms, with the production of acids. The feeding of lactose encourages the growth of those types of intestinal bacteria that predominate in the intestinal tract of breast-fed infants. The acid produced on fermentation of lactose apparently favors the absorption of calcium salts; however, with customary cow's milk formulas and with adequate vitamin D the utilization of calcium is ample for all

requirements regardless of the type of sugar added to the formula. In concentrated solution (10 per cent or above) lactose is somewhat irritating to the intestinal tract and may lead to an increased number of stools.

Sucrose

Sucrose (cane or beet sugar) has the same nutritive value as milk sugar. It is somewhat more quickly digested and absorbed from the intestinal tract and is not fermented as easily by most intestinal bacteria; it is, however, fermented by yeasts. Sucrose is not so laxative as milk sugar and may be fed with safety in somewhat larger amounts, especially to infants suffering from digestive disturbances. In concentrated solution, it is irritating to mucous membranes and may lead to vomiting and diarrhea. This is not true, however, of the more dilute solutions. Sucrose is the sweetest of the commonly used sugars. In general, sucrose is a suitable form of carbohydrate for the feeding of the average infant.

Products of Starch Hydrolysis ("Malt Sugars")

When starch undergoes digestion in the alimentary tract, it is converted first into intermediate products known collectively as the dextrins. These, by further hydrolysis, are converted into maltose and ultimately into dextrose.

When starch is subjected to the action of malt diastase, hydrolysis proceeds through these same stages as far as maltose. The relative proportions of dextrin and maltose in the final mixture will depend on the length of time the conversion is allowed to proceed: if for only a short time, there will be relatively large amounts of dextrin present; if for a longer time, the proportion of maltose will be greater. The properties of the various malt preparations depend to a considerable extent on the relative proportions of dextrin and maltose present. The source of the starch differs with the various commercial products.

When starch is heated appropriately and for a sufficient length of time, a small proportion is converted into dextrin.

Dextrinized starch, prepared in this manner, has found some use in infant feeding.

When starch is heated in the presence of acid, hydrolysis occurs through all the stages with the final formation of dextrose. This process is used extensively in the commercial production of corn syrup or "glucose," as well as in the production of corn sugar or dextrose. In the commercial production of corn syrup, corn starch is treated with mineral acid under steam pressure until a definite percentage of dextrose is formed. The product is then neutralized with sodium carbonate, filtered and concentrated. The final product contains dextrans, maltose and dextrose.

Dextrin

Chemically, dextrin is a mixture of a number of closely related substances. It is a white, or as usually obtained, slightly yellowish, somewhat sticky powder. It does not have a sweet taste, but is, if anything, slightly bitter. It is not very irritating, even in highly concentrated solutions, because of its low osmotic pressure. Dextrin is not fermented by most intestinal bacteria; it is, however, easily converted into maltose through the action of the enzymes normally present in the intestinal tract. This conversion does not appear to occur much more rapidly than the maltose produced can be digested and absorbed, so that when dextrin is fed there is not, at any one time, a large amount of readily fermentable carbohydrate present in the intestinal tract. Dextrin, therefore, is not likely to lead to intestinal fermentation, a factor which is an especial advantage in the presence of diarrhea. Dextrin may be given safely in larger amounts than other carbohydrates, and its value in nutrition is as great. Dextrin alone has not been widely used in infant feeding, mixtures of dextrin and maltose being the customary form. The amount of dextrin in these mixtures varies from a negligible amount to approximately 75 per cent.

Maltose

Pure maltose is not used in infant feeding, but mixtures of maltose and dextrin are extensively used. The proportion of maltose in these preparations ranges from 30 to 90 per cent. One commercial preparation contains less than 5 per cent dextrin. Maltose itself is a white powder, slightly sweet. It is easily fermented by most intestinal organisms, but at the same time is quickly converted into dextrose and absorbed, so that unless excessively large amounts are fed, serious fermentation is not likely to result. The products of fermentation of maltose do not seem to be as irritating as those of sucrose or lactose.

Dextrose (d-Glucose)

Dextrose, or d-glucose, requires no digestion, being immediately absorbable. Dextrose is very easily fermented, but is the most quickly absorbed of all the sugars commonly used. Fairly large amounts of dextrose may be added to the food of an infant without the danger of any great degree of intestinal fermentation. It is a valuable form of sugar in the treatment of the diarrheas. Dextrose is now available in a pure commercial form at a low price.

Dextrin-Maltose Mixtures*

Many dextrin-maltose mixtures prepared by the action of malt diastase on cereal starch are commercially available. Some of these are without salt or other additions; some have added sodium chloride, others added potassium carbonate or bicarbonate; some have added materials containing vitamin-B complex, chiefly extracts of wheat embryo and yeast. Some of the preparations are in dry powdered form, others are dispensed as syrupy liquids. A wide choice exists as to relative amounts of dextrin and maltose, with or without additions. These preparations

*For a list of these various sugars and their composition, reference should be made to "Accepted Foods and Their Nutritional Significance" published by the American Medical Association.

contain fair amounts of the vitamins of the B group, particularly thiamine, which are carried over from the grain.

The dry powdered preparations are in most common use. Among those without additions, it probably makes little difference which is chosen for the routine feeding of normal infants. Nutritionally they are equal. The differences in relative laxative effect due to different proportions of dextrin and maltose are unimportant.

The preparations with added potassium carbonate or bicarbonate are more laxative than those without such an addition. Some of the preparations owe their increased laxative effect entirely to the added potassium salt. Others have a laxative effect also because of extractives carried over from the grain mash; these latter products are less refined and are dispensed as heavy dark syrups, e.g. "malt soup extract."

Dextrin-Maltose-Dextrose Mixtures (Corn Syrup)

Two dextrin-maltose-dextrose mixtures are on the market, both prepared from corn starch by acid hydrolysis, both intended primarily for infant feeding. One is in a dry powdered form, the other is a syrup. On a water-free basis both products contain approximately 16 per cent dextrose.

Dextrin-Maltose-Dextrose-Sucrose Mixtures (Flavored Corn Syrup)

Corn syrup, or commercial "glucose," though used extensively in food manufacture, is not available in the retail trade, except for the two products mentioned in the preceding section. However, corn syrup flavored with either cane-sugar syrup or with refiners' syrup is available at every grocery in the country. The clear or "white" syrup contains 85 per cent corn syrup and 15 per cent cane-sugar syrup; the dark or "golden" syrup has the same composition, but is flavored with refiners' syrup instead of cane-sugar syrup. These two syrups are of equal nutri-

tional value. These table syrups are distributed under hundreds of individual brands, but all are of approximately the same composition. The composition, in general, is about as follows: dextrin, 50 per cent; maltose, 30 per cent; dextrose, 10 per cent; sucrose, 10 per cent. Many of the syrups are flavored also with vanilla or artificial substitutes. The composition of these syrups is such that one fluid ounce contains one avoirdupois ounce of carbohydrate. Though reference is often made to these products as corn syrup, such a designation is incorrect.

Mixed Sugars

A mixture of several carbohydrates possesses certain advantages over a single sugar, as larger amounts of such mixtures usually may be fed without the danger of producing diarrhea. At times the intestinal tract contains organisms which ferment certain carbohydrates more easily than others. When a mixture of carbohydrates is fed, only certain of the components may be fermented, so that excessive fermentation at any one time is not likely to occur. Thus there is often an advantage in such mixtures of sugars as have been discussed, when used as additions to milk which already contains lactose.

Molasses

Molasses is to be differentiated from table syrups prepared from corn syrup. Molasses is the mother liquor remaining after the crystallization of sugar in the process of cane sugar refining. It contains sucrose and invert sugar, together with extractives from the sugar cane. As marketed, it usually contains a small amount of sulfur dioxide. Molasses contains an appreciable amount of iron. The sugars of molasses ferment easily, and because of this property and of the extractives present, molasses has a distinct laxative effect and cannot be given in the same high concentrations as some other sugars. It can, however, be used satisfactorily for the feeding of normal infants, and is of value when there is a tendency to constipation.

Honey

Honey consists of a mixture of sugars, chiefly levulose and dextrose with some sucrose. In the intestinal tract it behaves much as has been described for dextrose. Honey, like other naturally occurring carbohydrates, contains some of the B group of vitamins. The amounts of these are unimportant in terms of the infant's requirement.

The Carbohydrates of Fruits

All fruits contain carbohydrates, largely dextrose and levulose, which are utilized readily by the infant. Of especial interest is the carbohydrate of bananas, which comprises about 22 per cent of the total weight of the ripe banana, or 85 per cent of the weight of the banana powder. Banana carbohydrate consists of a mixture of sugars, chiefly sucrose, dextrose, and levulose, and is very well digested and absorbed even by infants and young children with gastrointestinal disturbances. Ripe bananas have been used largely in the treatment of chronic intestinal indigestion (celiac disease). Dried banana meal has found some use in infant feeding. The feeding of banana leads to changes in the intestinal flora from gram-negative to gram-positive types, mostly *M. ovalis*. Bananas contain, besides carbohydrate, fair amounts of vitamins A, B-complex, and C. Only fully ripe bananas should be used.

Starch

Starch occurs as the chief constituent of all cereal grains and the tuberous vegetables. It has approximately the same composition, no matter what its source, but varies considerably in physical properties. Starch occurs in nature in small granules surrounded by envelopes of cellulose. On heating the grains, the cellulose envelopes rupture and the starch is liberated. Raw starch is digested with great difficulty because of the indigestibility of the cellulose envelope. Starch used for the feeding of infants

is prepared by milling the grain and subsequent cooking. Even when properly prepared and well cooked, starch is incompletely digested by the very young infant. If, however, starch is fed regularly from an early age, the infant gradually develops the power of digesting it fairly well, although at first much starch may be detected in the stools by the iodine test.

Starch does not ferment easily and acts, to some extent, as a demulcent in the intestinal tract. It is probable that the passage of undigested starch through the infant's intestinal tract does little or no harm in most instances, though occasionally it may give rise to slight fermentation and gas production.

Decoctions of starch, being colloidal, possess certain advantages as additions to infant diets. When starch gruels are added to raw cow's milk, the curds precipitated by the action of acid and rennin are more finely divided than would be the case otherwise. Another use of starch in infant feeding is in rendering milk mixtures so thick as not to be vomited easily. Such thick gruel feedings are used in the treatment of vomiting of various types, such as that due to pyloric stenosis or rumination. The starches chiefly used in infant feeding are those of barley, wheat, oat, corn, and rice flours. Of these, barley flour is the most widely used as it makes the most demulcent gruel.

During the process of digestion, starch is converted through the stages of dextrin and maltose to dextrose, in which form it is finally absorbed. It is not possible to meet the carbohydrate needs of the young infant by starch alone, but after the age of six months, starch may gradually replace the simple sugars in the diet.

Sugars as a Cause of Diarrhea

Any sugar, when fed in excessive amounts, may cause gastrointestinal fermentation and diarrhea. The sugars which have smaller molecules, such as the monosaccharides,

dextrose and levulose, and the disaccharides, lactose and sucrose, when in solution, have greater osmotic pressures than the higher carbohydrates, such as dextrin and starch. Concentrated solutions, therefore, act somewhat as hydragogue cathartics. Solutions of dextrose stronger than 6 per cent, or of milk sugar and cane sugar stronger than 8 per cent, are hypertonic and likely to exert some laxative effect for that reason alone, aside from fermentation. Dextrin and starch, on the other hand, may be given in solutions of a concentration of 20 per cent or more without being hypertonic. This is an important practical point in the construction of infant feeding formulas. It was, at one time, very generally stated that the total sugar in the final formula fed should not exceed 7 to 8 per cent. We now know that when sugars containing a considerable amount of dextrin are used, sugar concentrations up to 20 per cent may be given without harm and may at times even exert a constipating effect, because of the absorption by the intestine of large amounts of water along with the sugar.

Parenteral Administration of Carbohydrate

Sucrose, lactose, dextrin, and starch are not utilized when given parenterally, but dextrose is utilized when given intravenously, subcutaneously, or intraperitoneally. In the case of infants with lowered intestinal tolerance to carbohydrates, the parenteral administration of dextrose may serve to maintain the nutrition over critical periods. Dextrose solutions, when injected parenterally, also serve other important functions. The intravenous route is the preferable one. A moderate irritative effect is often observed when dextrose solution is given intraperitoneally.

CHAPTER V

FAT METABOLISM

Fats, like carbohydrates, are composed of carbon, hydrogen, and oxygen; the oxygen content, however, is low and much more oxygen is needed for the combustion of 1 gram of fat than for 1 gram of carbohydrate. All fats consist of a combination of fatty acids with glycerol.

Fats as such cannot be absorbed from the intestinal tract, but must first be saponified. Saponification consists in a separation of the fatty acid portion of the fat from the glycerin and is accomplished through the action of the pancreatic juice. The alkaline salts of the fatty acids are known as soaps. Fat, after saponification, is absorbed as glycerin and fatty acid (or as soap). These are reunited after passage through the intestinal wall to form neutral fat. There is, at the same time, a considerable rearrangement so that the recombined fat resembles more nearly the natural fat of the individual than the food fat. Fat is not absorbed directly into the blood, but is taken up by the lymphatics and carried to the blood stream in a fine emulsion by way of the lacteals and the thoracic duct.

The fat which enters the blood is in part carried to the subcutaneous tissues and deposited, and in part to the liver, where it undergoes a chemical transformation, rendering it capable of being metabolized as fuel. When there is relatively abundant carbohydrate and protein in the diet, but little fat is burned, the major part being stored. During starvation, however, a large portion of the energy requirement of the body is met by the utilization of fat, which is removed from the fat depots, carried to the liver by way of the blood and subsequently metabolized. The amount of fat used by the body, therefore, is dependent on the state of nutrition and the relative amounts of other

foods fed and not merely on the intake of fat. An infant who is starving may actually use larger amounts of fat than one who is fed a diet containing much fat with adequate amounts of carbohydrate and protein. The depot fat of the body constitutes an important reserve of food energy.

Formerly it was believed that fat cannot be metabolized completely by the human unless a certain amount of carbohydrate is used simultaneously; it was believed that carbohydrate is necessary for oxidation of fat beyond the stage of ketone bodies. It is the belief at present that incomplete oxidation of fat with resulting ketosis is the result of functional incapacity of the body to burn completely the large amounts of fat needed and presented for energy production when other sources of energy are inadequate, and that no other relationship exists between ketosis and the rate of carbohydrate utilization. The body normally uses carbohydrate for energy in preference to fat. Only when carbohydrate is not available is fat oxidized in large amount. Fat can be oxidized completely to carbon dioxide and water by the human, but only in limited amount at any one time; this amount approximates 2.5 grams daily for each kilogram of body weight for the adult. If fat must be oxidized in excess of this amount, oxidation is incomplete and the unoxidized residues, the ketone bodies, are excreted. The maximum rate of fat oxidation by infants is not known, but it is well below that of the adult. Prematurely born and immature infants develop ketosis more readily than full-term normal infants. For these reasons the infant, especially young or feeble infants, should derive the greater part of the energy requirement from carbohydrate rather than from fat.

Fat does not have the same essential function in nutrition as protein and carbohydrate and may, at least to a large extent, be replaced with carbohydrate as a source of energy. There are, however, certain constituents of fat which are essential and others which appear to be. Food fats are

carriers of certain of the vitamins. The important fat-soluble vitamin for the infant is vitamin A, of which milk fat is a good source. When the infant receives an appropriate formula of whole milk and the usual supplemental foods, the vitamin A requirement is met. For animals, unsaturated fatty acids, especially linoleic and arachidonic acids, have been shown to be essential; in the absence of these, the nutrition suffers and skin lesions develop. If a similar need exists for the infant, it would seem to be easily satisfied. Young children with chronic indigestion (celiac disease) have been maintained in good nutrition for long periods with extremely fat-poor diets, but with one teaspoonful of cod liver oil daily. A relationship has been thought to exist between deficiency of unsaturated fatty acids and infantile eczema. Such a relationship, however, has not been well substantiated. Observations indicating that animals fattened with a diet high in carbohydrate have less resistance to infection than those receiving a larger portion of food fat have not been well enough controlled for acceptance; it is probable that dietary deficiencies other than of fat were responsible for the differences observed.

When fat is omitted from the diet it becomes necessary to give larger amounts of carbohydrate and protein in order to meet the caloric need. In both human and cow's milk, fat provides approximately one-half of the total calories. In cow's milk formulas customary for the young baby, fat provides approximately 40 per cent of the calories. When infants are fed fat-poor diets, the stools tend to have a darker color; if the caloric requirement is satisfied largely with disaccharides, the stools at times may be "fermentative" in character.

Fat occurs in the milk of all mammals, but differs in composition in the various species. The amount of fat in average cow's milk as supplied by dairies is approximately the same as that in average human milk, viz., 3.5 to 4 per cent. In comparison with human milk, the fat of cow's

milk contains more of the esters of the lower fatty acids (butyric, caprylic, caproic, etc.). For the normal healthy infant this difference in the two fats seems of no importance. For sick infants who are likely to have gastric stasis and evidence of indigestion, the lower fatty acids apparently have an irritating effect, contributing to the occurrence or aggravation of vomiting and diarrhea. Cow's milk fat differs further from the fat of human milk in that it contains only about half as much of the unsaturated higher molecular weight acids, which include the so-called "essential fatty acids." The two milks contain approximately the same amounts of oleic, palmitic, and stearic acids. The fat of cow's milk is slightly less well utilized by the baby than is the fat of human milk, due chiefly to increased formation of relatively insoluble calcium soaps in the intestine because of the larger amounts of casein and calcium present in cow's milk. With the customary method of feeding, the difference in fat absorption from human and from cow's milk is nutritionally unimportant. When the amount of sugar is small in a cow's milk formula and the amount of casein (or milk) is large, constipation is the expected result, because of the greater formation of calcium soaps; the loss of fat, as soap, at the same time is greater. Overfeeding with fat may result in either constipation or diarrhea, depending on the relative amounts of casein and sugar present; increase in casein and decrease in sugar lead to constipation, the reverse leads to diarrhea.

In general, an excess of fat in the diet of the artificially fed infant should be avoided. Ordinary grades of cow's milk containing not more than 3.5 to 4 per cent of butter fat should be used in preference to the richer milk of Jersey or Guernsey cows. The relationship between fat and casein in ordinary grades of cow's milk is such that only in the presence of gastrointestinal disturbance or gross malnutrition is it necessary to reduce the fat content by partially skimming the milk.

The fat of milk exists in the form of a fine emulsion. There is in general no difference in the size of the globules in human as compared with fresh cow's milk, although there may be considerable variation in the milk obtained from different women. Cow's milk which has been homogenized, that is, atomized by passage through a valve under high pressure, has its fat in the form of globules approximately one-sixth of their original diameter. Globules of this size do not rise as cream when milk stands. Although a larger surface of the fat is exposed to the digestive juices, this change in physical state is of little importance in fat digestion, the completeness of digestion being independent of the size of the globules. The more definite effect of homogenization on protein digestion is discussed elsewhere.

Certain vegetable fats with a high proportion of unsaturated fatty acid are absorbed somewhat more readily than the animal fats more commonly fed to infants. The increased absorbability is of little importance for the normal infant, but may be advantageous for sickly and delicate infants. Removal of fat from milk and substitution of other fats would be an advantage in infant feeding only for frail infants and only if more readily absorbable fats were chosen. Some prematurely born, immature, and sick infants utilize any fat poorly. Failure of such an infant to gain while receiving an adequate calorie intake can often be traced to poor utilization of fat. In the case of many of these infants change in the type of fat fed does not alter the degree of utilization. Change of feeding to an isocaloric mixture of low fat content often results promptly in good gains in weight.

Fat is utilized when administered parenterally. A very fine emulsion may be given intravenously. However, animal experiments have indicated that this procedure may not be completely without harm and that this method of feeding should be reserved for emergencies only. In the animal experiments fat emboli were found in lung tissue

in 41 per cent four days after injection and in other organs in lesser degree. In some of the animals the fat emboli caused gastric and duodenal ulcers.

In the metabolism and storage of fat, less water is required and less is stored than in the case of carbohydrate. The storage of carbohydrate requires a concomitant storage of much water; consequently carbohydrate is a hydrating food. While fat is not to be considered as having a dehydrating effect per se, it does have such an effect practically, when it is substituted for carbohydrate in the diet. Advantage is sometimes taken of this relationship by prescribing a high-fat or ketogenic diet for those past infancy, but the occasions for practical application in infancy are few.

CHAPTER VI

MINERAL AND WATER METABOLISM

Inorganic Constituents of the Body

The human body contains the following elements: oxygen, carbon, hydrogen, nitrogen, calcium, phosphorus, potassium, sulfur, sodium, chlorine, magnesium, iron, iodine, copper, and minute amounts of several other elements (manganese, zinc, fluorine, silicon, and probably others), some or all of which may be necessary. The first four elements named (O, C, H, N) are present in largest proportion, as all body substance contains them. Calcium, phosphorus, and magnesium are a part of the structure of bone. Phosphorus and potassium are a part of the composition of cell protoplasm. Sodium and chlorine have a highly important function as essential constituents of interstitial fluid. Sulfur is a part of the protein molecule. Iron plays an important role in the formation of hemoglobin and muscle tissue. Iodine is necessary for thyroid function. Both iron and iodine are in organic combination in the body, and are excreted, if at all, as inorganic salts. Other elements in addition to sulfur, iron, and iodine also exist in the body both as organic and inorganic salts. The relative amounts of the various inorganic salts present in the body of the infant differ considerably from those of the adult because of the higher proportion of body water in the infant and the lesser degree of calcification of bone.

Some of the inorganic salts are constantly excreted and replacement from food must be continuous. The food normally received by the infant may be expected to contain, with the possible exception of iron and iodine, an adequate amount of all the necessary minerals and a considerable excess of some. Any excess that may be present in an ordinary diet is excreted promptly without harm. When

the diet is deficient in the various minerals or when they are lost in large amounts by vomiting, diarrhea, or failure of absorption, pathological states become evident, the outstanding condition present being dependent on the material predominantly lacking. Thus there may be interference with growth and cell function in general; or rickets, tetany, acidosis, alkalosis, or anemia may develop. Mineral salts are necessary for the digestion of food and for the maintenance of osmotic relationships and interchange between cells and body fluids.

Body Water, Sodium, Potassium and Chloride

Each cell of the body contains some water; in addition, large amounts of fluid are in the spaces between the cells and in the channels for fluid distribution. Water within the cells is a part of the cell protoplasm and is spoken of as intracellular water; that outside the cells is called extracellular water and includes water of plasma and lymph, as well as that of the fluid bathing the individual cells, the latter often being called interstitial fluid. The compositions of extra- and intracellular fluids differ. Interstitial fluid is of fairly constant composition and contains almost all the body chloride and about three-fourths of the body sodium. Sodium chloride and bicarbonate are the chief salts, with small amounts of potassium, magnesium, and calcium. In general, body cells, with the exception of the red cells of the blood, contain practically no chloride or sodium, but contain most of the potassium of the body, combined with phosphate and protein. Thus, as body tissue increases, potassium and phosphate are needed as well as protein. As the body content of extracellular water changes, alterations in sodium chloride content parallel those of the water.

The water content of a normal newborn infant has been compared with that of an edematous adult. In the normal adult about 20 per cent of the body weight is due to extracellular water, in contrast to 40 to 45 per cent of the weight

of the newborn infant from this source. As the extracellular (interstitial) water is the "labile water" of the body, it is easy to understand the sudden and marked losses of water that occur during even comparatively mild illnesses during infancy. It seems obvious that to replace this interstitial fluid, the ions it normally contains must be replaced also. Sodium chloride is the largest component of the salts of interstitial fluid. In severe vomiting or diarrhea, chloride losses (from vomited gastric juice) or losses of base (from intestinal juices) may be more vital than the water losses. These losses may lead to important changes in the acid-base balance, either in the direction of alkalosis or of acidosis, depending on whether the predominant loss is of anion or cation.

As long as an infant is ingesting milk—either human milk or customary formulas of cow's milk—and is retaining it, the intake of sodium, potassium and chloride will be ample, with some to spare. No additions of these materials to the diet are necessary, though in moderate amount they do no harm to the normal baby because the excess is promptly excreted in the urine.

The water requirement of the infant in proportion to his body weight is high, being approximately three times that of the adult. The high water requirement of the infant is to be explained on the basis of the more active metabolism and less efficient kidneys. The infant's heat output is greater in proportion to his weight than that of the adult, and heat is removed from the body chiefly by water evaporation. The relatively large food intake requires constant circulation of water from the blood to the intestinal tract and back into the blood during the processes of digestion and absorption. Additional large amounts are required for the urinary excretion of waste products. An infant cannot excrete as concentrated urine as an adult. The average daily urinary excretion of the normal infant, fed in a customary manner, is from 50 to 75 ml. for each kilogram of body weight; prematurely born infants have

still smaller ability to concentrate urine and may excrete more than 100 ml. for each kilogram with an intake of approximately 170 ml. for each kilogram.

Of the total water intake under normal conditions, approximately 40 to 50 per cent is eliminated by the kidneys, 40 to 50 per cent by evaporation from the skin and lungs, and 5 to 10 per cent by way of the bowel. Approximately 3 per cent of the water taken by the young infant is retained by the body; this amount represents from 55 to 70 per cent of the total weight gain. The amounts eliminated by the various channels are greatly influenced by extraneous conditions. In the presence of high external temperatures, an enormously increased elimination of water from the skin is necessary in order to maintain the normal body temperature. Vigorous exercise also increases markedly the water loss from the skin and lungs. These losses are at the expense of urine water. In the presence of severe diarrhea, the water loss from the bowel may equal or exceed the fluid intake. In severe vomiting, much of the water taken by mouth may be lost. In any condition associated with an increase in the rate or volume of the respirations—for example, in pneumonia or acidosis—the water elimination from the respiratory tract is increased.

In the light of the preceding considerations, it is difficult to state specifically the water requirements of the individual infant since this is subject to considerable variation. The average normal breast-fed infant under six months of age receives from the milk approximately two and one-quarter ounces of water daily for each pound of body weight (150 c.c. for each kilogram). This amount suffices to meet the needs of most infants and to allow a reasonable excess, except during extremely hot weather or in the presence of diarrhea or vomiting. In the case of artificial feeding, the protein and salt content of the diet is usually higher than with breast feeding; consequently increased elimination of salts and nitrogenous waste products occurs in the

urine, which requires a somewhat greater fluid output by the kidneys. The extra amount required, however, is slight, and for practical purposes the artificially fed infant, under average conditions, need be given no more fluid than the one fed at the breast. It is not necessary that the total fluid given be contained in the milk or milk mixture fed. Water may be fed from a bottle or cup and, under special conditions, fluid may be given as dextrose and salt solutions subcutaneously, intraperitoneally, or intravenously.

An insufficient water intake or excessive loss of water from the body is incompatible with a normal gain in weight. In extreme cases in which the water output actually exceeds the intake, rapid loss in weight occurs together with the development of serious symptoms which may eventuate in death. The correction of this type of difficulty involves the administration not only of water but also of chloride in order that the water may be retained.

A reasonable excess of water intake over the actual requirements produces no serious disturbance, but great dilution of the food is undesirable, as this necessitates the intake of a large volume in order that the infant may receive sufficient of the food constituents. Not infrequently the infant's stomach capacity is insufficient to permit the large intake required when dilute formulas are used. One of the all-too-common errors in infant feeding is the giving of too-dilute formulas, based on the idea that the individual food components cannot be digested and absorbed when present in the formula above certain concentrations. There are, of course, limits to which the concentration of a formula may be carried, but most infants can take—and can thrive on—considerably more concentrated formulas than was at one time supposed. In general, it is not necessary that the volume of the formula should exceed 10 per cent of the body weight. Occasionally, however, in the case of undernourished infants, it is necessary to give 50 per

cent more than this in order to supply the necessary food constituents even when fairly concentrated formulas are used.

Calcium, Phosphorus, and Magnesium

As more than 98 per cent of the body calcium is in the skeleton, any discussion of the calcium metabolism resolves itself largely into a discussion of skeletal growth and rate of mineralization. The skeletal mineral is a calcium-phosphate-carbonate complex of somewhat variable composition. A small part of the bone calcium may be replaced by magnesium and sodium. Very small amounts of other minerals and anions also are present. The exact process involved in the formation of bone salts is not wholly understood. The available amounts of calcium and phosphorus and of vitamins A, C, and D are important factors, as also are certain of the endocrine secretions. Of the latter, the secretions of the parathyroid, thyroid, pituitary, and sex glands all have some effect on the metabolism of bone. In infancy, disturbances of bone metabolism are more often due to vitamin deficiency than to endocrine disturbance.

Mineralization of the skeleton is very active during the last month of fetal life. At birth the entire body of the average normal full-term infant contains about 25 grams of calcium. If the mother's diet has been extremely poor in calcium, phosphorus, and vitamins C and D, the amount of calcium in the body of the infant will be less than normal. A deficiency of sufficient degree to produce fetal rickets has been reported only for infants born of osteomalacic mothers. From recent studies it appears that poorly nourished women give birth to small infants.

At birth the bones of the infant contain much more water and less mineral than do the bones of an adult. After birth there appears to be a period of adjustment wherein the rate of mineralization does not keep pace with the rate of growth of the skeleton. This period of adjustment lasts somewhat longer in breast-fed infants than in those babies

receiving the larger amount of calcium and phosphorus in cow's milk. Human milk contains about 300 mg. of calcium to the liter. The breast-fed baby excretes very little calcium in the urine, but the minimum fecal output seems to be about 150 mg. daily. Thus a young baby taking only 600 ml. (20 ounces) of human milk a day could retain as a maximum only about 50 mg. of calcium daily. By the time the baby is three months old, the milk intake is sufficient to permit more rapid deposition of minerals, and the percentage of calcium in bone increases steadily.

The prematurely born infant labors under a double disadvantage as far as calcification of the skeleton is concerned. A baby born four weeks prematurely has a calcium content only a little more than one-half that of the normal full-term infant. In addition, his food capacity is limited. It is difficult to feed sufficient human milk to permit any calcium retention. The rapid growth and very slow rate of calcification may result in early and severe rickets. The practice of fortifying human milk with dried skimmed milk for prematurely born infants permits sufficient intake of usable mineral to prevent rickets, provided that vitamin D also is supplied.

The infant given cow's milk ingests an ample quantity of both calcium and phosphorus, as cow's milk contains about 1200 mg. of calcium and 900 mg. of phosphorus to the liter. Without vitamin D the calcium and phosphorus of cow's milk are poorly utilized by the infant. If vitamin D is provided in adequate amounts (300 to 400 units daily), the absorption and retention of calcium and phosphorus from cow's milk are considerably higher than those of the breast-fed infant. The urinary excretion of calcium by the infant fed cow's milk varies from 5 to 20 mg. daily for the young baby and averages about 20 to 30 mg. daily for the older infant. The young baby receiving cow's milk retains daily approximately 50 mg. of calcium for each kilogram of body weight.

Illness, even slight and afebrile, decreases the mineral retention of infants markedly and leads to a temporary slowing of growth, even in the well-fed infant. For the infant not given sufficient vitamin D, such illnesses are undoubtedly factors predisposing to the development of rickets.

The amount of calcium in body fluids is quantitatively small but highly important in regulating the irritability of the tissues. The serum calcium is maintained normally at 10 to 12 mg. for each 100 ml. (5 to 6 milliequivalents to the liter). In infantile rickets the serum calcium is usually near the lower normal level. If the healing of rickets is interrupted, the serum calcium level may fall abruptly to as low as 6 mg. in 100 ml. and the phosphorus level rises. Active tetany may be expected to occur whenever the calcium level decreases below 7 mg. and may occur with a higher level if the serum phosphorus is much increased. (See Chapter XXX for further discussion of mineral relationships in tetany.)

When babies are fed cow's milk, in the customary quantities and manner, a need for additional amounts of ingested calcium probably never arises, since the quantity of calcium in milk is ample for all purposes. Low blood calcium values, when observed in infants, are produced by poor absorption because of vitamin-D deficiency. Additions of calcium to the diet do not improve calcium utilization. Calcium salts, when administered parenterally in infantile tetany, give desirable but very transitory relief. When calcium salts are fed by mouth or injected parenterally in these circumstances, the calcium is excreted chiefly by way of the bowel and the anion, or acid component of the salt, is excreted by way of the urine. The administration of calcium chloride is therefore, to a certain extent, equivalent to the administration of hydrochloric acid, since the acid component must be neutralized and excreted. The administration of mineral acid serves to ameliorate the symptoms of tetany, even without increase in the calcium

content of the blood. The organic calcium salts—the lactate, acetate, and gluconate—act as bases, the acid components of these salts being metabolized to carbon dioxide and water.

At times, chiefly because of allergy, it becomes necessary to feed babies a diet devoid of milk. In these circumstances, calcium salts must be added to the diet. Such proprietary foods as are available for this purpose already have the needed calcium additions. Whenever calcium is added to the diet for the purpose of maintaining bone growth, it is necessary to keep in mind that calcium is not utilized unless appropriate amounts of phosphorus are available at the same time. Though most of the protein-containing materials used as the basis for milkless diets contain some phosphorus, the amount of phosphorus present may not be adequate for calcium utilization. Consequently, whenever calcium salts are ingested for the purpose of maintaining bone growth, at least a generous proportion should be in combination with phosphorus. Either di- or tri-calcium phosphate serves the purpose well.

Phosphorus is used in the body not only in the bones, but also in the soft tissues: it is an essential part of the skeletal mineral, of the phospholipids of the nervous system and other tissues, of the protein of the cell nucleus, and of nitrogenous compounds of intracellular fluids. The quantity needed depends both on the intake of calcium and the intake of protein. Human milk has a relatively low content of both calcium and protein; therefore, the amount of phosphorus needed is relatively small, and in this food the ratio of calcium to phosphorus of 2:1 suffices. When a diet higher in protein is given and the protein retention is correspondingly greater, the phosphorus intake must be increased also. If cow's milk is given, the increased need is automatically met, as cow's milk contains ample phosphorus for its calcium and protein. In formulating special foods such as are discussed in the preceding paragraph, it is an error to consider that either the quan-

tity of calcium and phosphorus, or their ratio to each other, will be optimum when they are the same as in human milk. These foods are usually fed in such a manner that the nitrogen retention is as high as with cow's milk feeding, and if the phosphorus intake is not ample, insufficient phosphorus will be available for bone mineralization. In all artificial food mixtures the phosphorus content should be related to both the calcium and the nitrogen content of the food.

Magnesium, like phosphorus, is used both in bone and soft tissue. It is an essential constituent of cell fluids, though the amount so used is less than that deposited in bone. The quantity of magnesium needed by the infant is not certainly known. It may be assumed that human milk contains sufficient to meet the requirement when the baby is breast fed. Like phosphorus, magnesium is required in larger amounts when cow's milk or a synthetic food is fed. Cow's milk contains more than twice as much magnesium as does human milk and the amount present seems to be ample. Calcium and magnesium are antagonistic in some respects and complementary in others. Calcium promptly overcomes the respiratory depression produced by magnesium, but salts of both these elements relieve tetany.

Sulfur

Sulfur is a constituent of certain of the amino acids of protein, and presumably it is in this form only that it is available to the body. In the body sulfur is similarly a component of protein. It is a constituent of insulin and of glutathione of red blood cells and muscle. Glutathione acts as a coenzyme concerned with the metabolism of carbohydrate.

Methionine is the one essential amino acid that contains sulfur. However, cystine, also sulfur-containing, can substitute for methionine to a certain extent. Casein contains more methionine than cystine. The sulfur contents of hu-

man and of cow's milk are 0.014 and 0.031 per cent, respectively; less of the sulfur of human milk is protein sulfur than is true for cow's milk. Infants retain about 90 per cent of the sulfur intake and the remainder is excreted in the urine. The sulfur of infants' urine is largely unoxidized or "neutral" sulfur, whereas older children and adults oxidize a large part of the waste sulfur to sulfate before excreting it.

Iron

Iron is a constituent of all the cells, but it is present most abundantly in hemoglobin of blood and muscle. The iron requirement of the infant is relatively high because of the rapid rate of formation of new body tissue. The diet of the young infant contains an amount of iron that is negligible in relation to his need. The chief source of iron for the young infant lies in the excess of hemoglobin present at birth. The low oxygen tension of the placental circulation results in a high hemoglobin concentration in the red blood cells of the fetus. At birth the number of red cells for each unit volume of blood is approximately the same as in adults, but the hemoglobin concentration is much greater, varying from 15 to 25 grams for each 100 ml., and averaging about 22 grams. The highest concentrations are obtained when the cord is not tied until pulsation ceases. As soon as the lungs begin to function, the high hemoglobin concentration is unnecessary. The excess is broken down, but most of the iron is stored in the liver and used later. If the mother's iron intake is ample during pregnancy, an added 50 to 100 mg. may be stored in the fetal liver before birth.

Destruction of excess hemoglobin continues to the ninth or twelfth week of life. At the final minimum level the hemoglobin values range from 10 to 12 Gm. to each 100 ml. A slight secondary rise then occurs, due to the stimulus of the stored iron, so that by the fifth month the hemoglobin values range from 11.5 to 14 Gm. to each 100 ml. of

blood and remain at this level throughout infancy if the intake of iron is adequate. After the fifth month, however, if additional iron is not supplied in the food, the stored iron is insufficient to meet the body needs and the amount of hemoglobin decreases gradually throughout the remainder of infancy. Illness, either infectious or gastrointestinal, is followed by further decreases in hemoglobin. Thus, unless special precautions are observed, an infant is liable to develop secondary anemia during the latter half of the first year.

The baby born prematurely is smaller and contains less blood than the average full-term infant. The prematurely born infant, therefore, has less excess hemoglobin to break down and smaller amounts of iron to be stored for future use than does the larger baby born at term. Furthermore, prematurely born babies are expected to grow at a rate sufficiently rapid to become equal in size to the full-term baby at a relatively early age. Infants born prematurely can thus be expected to become anemic earlier than infants born at term unless additional iron is given.

Feeding additional iron during the period of hemoglobin destruction does not result in iron retention unless large amounts are given. Some evidence exists that prophylactic feeding of small amounts of iron may shorten the period of hemoglobin destruction and prevent the excessive lowering of blood hemoglobin often observed in prematurely born infants during this period. In general, however, there seems to be little advantage in additional dietary iron before hemoglobin destruction has ceased. Though the stores of iron in the full-term baby may be expected to last until he is five or six months of age if he has been in good health during this period, it does not seem wise to await complete depletion of the stores before supplying additional amounts. At least some reserve in storage should be considered normal. An allowance of 1 mg. of iron daily for each kilogram of body weight seems to permit ample retention; this would mean a total of approximately 6 to 7

mg. daily at six months, and 10 mg. at one year. It has been stated that for maximum hemoglobin levels, 25 mg. of iron should be given daily; such an intake is impossible from foods of the infant's dietary and must be supplied by adding iron salts to the feeding. Such a large intake of iron does not seem necessary. In order that retained iron be utilized it is necessary to have adequate intakes of copper and vitamin C and probably also of thiamine.

Both cow's and human milks contain very small amounts of iron. Human milk contains from 0.4 to 1.4 mg. to the liter; cow's milk from 0.15 to 0.6 mg. to the liter. The iron of human milk is better utilized than is that of cow's milk. The iron content of cow's milk may be much increased if it is kept in iron containers. Milk, which has been kept for several hours in ordinary milk cans, may contain as much as 2 mg. to the liter. It is obvious that the amount of iron in milk is wholly inadequate to supply the infant's requirement. For this reason it has become customary to begin feeding iron-containing foods at the close of the period of hemoglobin destruction (three months of age) or soon thereafter. Egg yolk and proprietary fortified cereal foods are the most commonly employed sources for initial additions. An egg yolk adds 0.5 to 1.5 mg. of iron to the day's intake, depending on the size of the egg and the food of the hen. Highly refined cereals may contain only 0.7 mg. of iron to 100 Gm. (0.2 mg. to the ounce), while proprietary cereal foods fortified with iron may contain as much as 17.5 mg. to the ounce. Subsequent additions, at the age of four or five months, consist of puréed vegetables and fruits. Vegetables as fed contain from 0.2 to 0.7 mg. of iron to the ounce (30 grams), fruits 0.1 to 0.3 mg. The vegetables and fruits, even though not rich sources of iron, average a higher content than refined cereals. The iron of certain of the vegetables, for instance, spinach, is not easily available to the baby.

Infants utilize iron salts very well. Ferrous sulfate and ferric ammonium citrate are the two most commonly used.

The ferric salts are probably reduced to ferrous salts in the gastrointestinal tract. The requirement of 10 mg. at one year would be supplied by 60 mg. of iron and ammonium citrate (1 grain), or 10 ml. of a 1 per cent solution. Such a solution can be added to the milk without altering the taste. Larger amounts of iron are useful temporarily in the management of iron-deficiency anemia. It is inadvisable, however, to give more than 10 mg. of iron daily for each kilogram of body weight; this is the equivalent of 250 to 300 mg. (4 to 5 grains) of one of the iron salts mentioned, for a baby of 4.5 kilograms (10 pounds) weight. Larger quantities of iron not only give no increased benefit, but they may be definitely detrimental and often cause gastrointestinal disturbances.

Copper

Copper is needed for the utilization of stored and absorbed dietary iron in its conversion to hemoglobin. The amount needed is small. The ratio of 1 mg. of copper to 10 mg. of iron is commonly considered as optimum in increasing the hemoglobin level of the blood. If the iron requirement of the infant is placed at 1 mg. for each kilogram of body weight, the daily copper requirement would be 0.1 mg. A small amount of copper is stored in the liver at birth. Human milk contains from 0.21 to 0.28 mg. to the liter, an amount which supplies approximately 40 micrograms for each kilogram of body weight. Fresh cow's milk contains from 0.09 to 0.21 mg. to the liter, an amount that might be inadequate in the early months were it not for the liver store. In the commercial handling of cow's milk (pasteurization, evaporation, drying), exposure to copper alloys permits increases in the copper content, which is often raised to that of human milk, or higher. The supplementary foods used in infancy contain minute amounts. In general, it appears that the amount of copper in the baby's food, though small, is adequate for his needs.

Except in the presence of anemia, the addition of extra copper to the diet other than that supplied in the natural foods is of questionable value. Furthermore copper has a distinct toxic action and the effects are cumulative. In the presence of anemia, when iron salts are administered, recovery may be hastened by the addition of a small amount of copper for a short period of time. Commercial iron salts used in medication usually contain some copper, though not the 10 per cent desirable for iron utilization. Inasmuch as the maximum amount of iron given to a baby with anemia should not exceed 10 mg. for each kilogram of body weight, the amount of copper should not exceed one-tenth of this amount, or 1.0 mg. for each kilogram. In terms of copper sulfate, 1.0 mg. of copper corresponds to 4 mg. of the salt, or 0.4 ml. of a 1 per cent solution.

Iodine

Iodine is necessary for normal functioning of the thyroid gland. At birth the thyroid of a normal infant weighs from 1.5 to 2 grams. In iodine deficiency the size of the gland is increased up to 10 grams or more. Thyroxin is formed in the normal fetal thyroid some time before birth.

The iodine requirement during infancy is not yet known, but it must be small, as the requirement for adults is stated to be 0.002 to 0.004 mg. for each kilogram daily. Iodine deficiency of the mother affects the fetus, the degree of hypothyroidism in the infant paralleling the amount of deficiency in the mother. In regions where goiter is prevalent, the use of iodized salt by pregnant women has decreased the incidence of cretinism markedly. The higher iodine content of the iodized salt of this country (1 part of iodide to 10,000 parts of salt) is more efficient in prophylaxis than the lower content used in Switzerland. Often additional iodine, 10 mg. weekly, is given to nongoitrous pregnant women in districts where goiter is common.

The iodine content of both human and cow's milk is variable, depending entirely on the iodine content of the

food. In regions where goiter is prevalent, the soil and water are poor in iodine and the iodine content of milk (as of other foods) may be negligible. On the other hand, near the sea coast, the iodine content of cow's milk may be from 1.5 to 2 mg. to the quart— or more than 100 times as great as the lowest values reported for other regions. Human milk varies in iodine content from 0.02 to 0.15 mg. (20 to 150 micrograms) to the liter. Of the supplementary foods given to the infant, cod-liver oil is the best source of iodine. A teaspoonful of cod-liver oil adds 12 to 50 micrograms to the day's intake. The iodine content of egg yolk varies with the iodine intake of the hen. Of the other foods, in any given locality the green vegetables, milk, and fruit usually contain more iodine than the cereals grown in that locality.

It appears from experiments with rats that 50 to 100 times the minimum protective dose of iodine does not affect the rate of growth or result in any other signs of toxicity. Infants receiving from 5 micrograms up to a maximum of 600 micrograms, daily, remain apparently normal. It appears that the iodine needs of the infant who is receiving one teaspoonful of cod-liver oil daily are adequately met. A more definite statement of requirement must await further investigation.

Fluorine

The fluorine of the body is chiefly in the bones and teeth. The fluorine content of bone tends to increase with the age of the person. Fluorine appears to be essential for good tooth calcification, as carious teeth have a lower content of fluorine than sound teeth. The fluorine ingested in foods is utilized much less than that in water. The presence of fluorine in drinking water in an amount of about one part per million during the period of calcification of the teeth produces harder teeth that are more caries resistant than the average for this country. Ingestion of

such water after the teeth have erupted seems also to have a favorable effect on resistance to dental caries, though fluorine is only one of several factors important in determining the resistance of the teeth. An excessive amount of fluorine, 1.5 to 2 parts per million or more, in drinking water during the period of tooth calcification, leads to the formation of soft chalky enamel that discolors and erodes easily. This condition is designated as mottled enamel. Such teeth are somewhat more resistant to decay than teeth with low fluorine content, but decay, once started, is devastating because the chalky material will not hold fillings well.

Manganese

Evidence is accumulating that manganese is an essential element for growth, development, and reproduction of many species. It seems essential in bone formation and for functioning of certain enzyme systems. Changes in the bones of young animals suffering from manganese deficiency differ from those of rickets and may be due to suppression of osteogenesis. It appears that other factors of the diet, calcium, phosphorus, and the vitamins, influence the degree of utilization of manganese by animals and birds. As many soils are deficient in manganese, the possibility of human deficiency must be presumed, although such deficiency has not yet been recognized.

Cobalt

Cobalt has been found important in hemoglobin formation in certain animals, but no need by the human has been shown. Any trial use of cobalt should be attended with great caution because of possible toxic effects.

Zinc

Zinc is widely distributed in animal and plant tissues, and is essential for the nutrition of plants. The human body contains nearly as much zinc as iron. It is found

in red blood cells, in the pancreas, spleen, liver, and kidney. Supposedly zinc functions as an integral part of certain enzyme complexes, as carbonic anhydrase and insulin. Apparently little danger of zinc deficiency exists in this country because of its wide distribution in food plants.

Acid-Base Balance

In the ash or mineral residue of both human and cow's milk, basic constituents are slightly in excess of acid. In most fruits and vegetables the excess of base is more marked than in milk, whereas in cereals, eggs, and meats, acid constituents predominate. In the infant's ordinary diet there is not a sufficient excess of either acid or base to be of any special significance. Consequently the acid-base equilibrium of the infant ordinarily requires no attention during health. Departures from the normal acid-base balance as they occur in certain illnesses require special management and are discussed elsewhere.

CHAPTER VII

THE VITAMINS

The vitamins are organic compounds, needed by the body in minute amounts, which the body is unable to synthesize, although certain of them may be synthesized in small amount by bacteria in the intestinal tract under favorable circumstances. Their grouping under one classification is largely due to historical influence, as chemically they have less in common with each other than with certain other chemical compounds in the body.

Knowledge of the chemical nature and physiological effect of the vitamins has grown with increasing rapidity each successive year. Many have now been isolated and synthesized. With the advent of vitamins in pure form, the physiological effect of each has been more accurately determined, and knowledge of the normal requirements is becoming more precise.

It is now known that two or more compounds closely related chemically may exert a similar vitamin effect. Thus there are several compounds with vitamin D activity, several with vitamin K activity.

A common, though artificial, division of the vitamins separates them into two classes: one soluble in fat and consisting of vitamins A, D, E, and K, the other soluble in water and including the B complex and vitamin C. This division according to solubility is not precise, for some compounds with vitamin K activity are water-soluble. It is, however, a convenient designation, inasmuch as two or more fat-soluble vitamins often occur together, and several members of the vitamin-B complex, especially, are found in the same extract.

Vitamin A

Vitamin A exists in two chemical forms known as A_1 and A_2 . Other forms of vitamin A may occur, but none has yet been isolated. Vitamin A activity is possessed also by certain carotenoids which are converted to vitamin A in the body. Those carotenoids with vitamin A activity that are of interest in human nutrition are alpha, beta, and gamma carotene, and cryptoxanthine. These pigments give the yellow color to many fruits and vegetables and are also found in green vegetables in association with chlorophyll.

Vitamin A_2 seems to occur only in fresh-water fish, where it exists together with vitamin A_1 ; it seems to have vitamin A activity, but little is known of its relative value. Vitamin A_1 , the familiar colorless form found in animal products (milk, eggs, fish oils, especially fish-liver oils), is easily utilized by human beings at all ages. The provitamins are less efficiently utilized even by adults, and presumably infants are still less efficient, although it has been reported that almost 40 per cent of the carotene ingested by the baby is absorbed.

It seems to be established that the esters of vitamin A (as found in naturally occurring products, such as fish-liver oils) have a greater biological activity and are more stable than the free form, which is found in the nonsaponified fractions of these oils. In other words, the vitamin A of the original oil is more stable and better utilized than the form in the concentrate of these oils.

Vitamin A is essential for body growth and for the normal status of epithelial structures. It is important for orderly bone growth and is necessary for normal metaphyseal activity. It is a component of visual purple and visual violet, retinal pigments responsible for the ability to see in a dim light. Deficiency of vitamin A in animals during early pregnancy leads to malformations of the fetus with defective eyes and other structures. Vitamin A has been

known in the past as the anti-infective vitamin, but such a designation is not applicable to vitamin A more than to other vitamins, except in so far as epithelial changes in the conjunctiva and respiratory tract resulting from vitamin A



Fig. 5.—Xerophthalmia, the result of a diet deficient in vitamin A.

deficiency predispose to secondary invasion by bacteria. The changes in the conjunctiva, when in an advanced state, give rise to the condition known as xerophthalmia. Among the earliest of the clinical demonstrable changes resulting from vitamin A deficiency are those affecting dark adaptation, or the ability to see in a dim light.

The vitamin A of both human milk and cow's milk occurs as a mixture of vitamin A and carotene, the total amount and distribution depending on the diet in the case of human beings and on the feed and breed in the case of cattle. The total amount in cow's milk varies from 22 to 90 international units to each fluid ounce, or from 730 to 3000 units to the liter, depending on the season and the type of feed. Winter milk in this country averages 36 units to the fluid ounce or 1200 to the liter, and summer milk 56 units to the fluid ounce or 1875 to the liter. The vitamin A content of human milk varies widely among individual women. In this country and in Europe the average range of values reported for human milk seems to be within the range found for cow's milk. It has been reported that for certain districts in the Far East human milk may contain as little as one-third the average amounts reported for this country. It is quite possible, then, that a woman ingesting a very restricted diet will secrete milk containing little vitamin A. Colostrum, both human and bovine, is very rich in vitamin A, containing from 2 to 10 times the amount found after lactation is established. The amount of colostrum secreted is so much less than that of milk after lactation is established that the day's intake of the vitamin from colostrum is usually lower than the intake after lactation is established.

Vitamin A and the carotenoids are absorbed from the small intestine after hydrolysis, and are re-esterified in the intestinal wall with either the fat carrier or other fatty acids. The vitamin appears to be absorbed more rapidly from unsaturated than from saturated fats, and the rate of absorption seems to be increased when lecithin is fed. The peak of absorption occurs three to five hours after ingestion. Absorption is increased in the presence of bile. Transfer through the body occurs both through the lymphatics and in the blood stream. The absorption of carotene is much less efficient in infants than that of preformed vitamin A. Carotene is easily soluble in mineral oil and is

carried from the intestinal tract by the oil when it is used as a laxative. Vitamin A is somewhat less soluble in mineral oil than carotene. Preferably mineral oil should not be given to infants or small children, and at any age it should be given out of relationship to meals.

Any disturbance of fat absorption will affect the absorption of vitamin A, as well as of other fat-carried vitamins. Celiac disease, cystic disease of the pancreas, hepatitis, and obstruction of the bile ducts are associated with poor absorption of vitamin A. Absorption is decreased in acute and chronic infection. In diarrheal diseases absorption of all food components, including vitamin A, is decreased. Impairment of absorption of vitamin A has been reported for an infant with intractable eczema.

Vitamin A can be stored in relatively large amounts in the liver and a goodly store is to be considered normal and desirable. In general, the storage is in proportion to intake and utilization. During illness and in certain abnormal states absorption is impaired and the stores become depleted.

The plasma vitamin A of newborn infants seems to bear no consistent relationship to that of the mother. The reported range is wide, namely, from 24 to 167 units for each 100 ml. for infants and 48 to 170 units for each 100 ml. for the mothers. The plasma vitamin A tends to fall for the first few days after birth, then rises as feeding is established. The plasma vitamin A of older infants is within the range for adults, but prematurely born infants usually show a low plasma level and poor liver storage. In general, a high level of plasma vitamin A indicates some storage and a low plasma level indicates poor or no storage. These relationships may not hold for the individual person, however.

The carotene content of the serum of newborn infants has been found to be low (0 to 13 micrograms for each 100 ml.), whereas the values for the mothers varied from 16 to 230 micrograms for each 100 ml.

At one time it was thought that a relationship existed between the amount of vernix caseosa in the newborn and the level of vitamin A in plasma, but no correlation appears to exist.

The vitamin A requirement of young mammals has been studied by the dark adaptation technique and by examination of blood levels. By the former technique apparently all warm-blooded animals studied, including human beings, have approximately the same requirement for each unit of body weight. It is generally agreed that the requirement is in relation to body weight. Current recommended allowances for vitamin A are based on the concept that the requirement of the young is greater in proportion to weight than that of the mature. The evidence for a greater requirement is not on a satisfactory basis, though apparently the young show evidence of deficiency more quickly than do the mature. The minimum requirement for the adult is presumably the same as that for the infant and has been estimated at 20 units (4 micrograms) as vitamin A or 40 units (25 micrograms) as carotene for each kilogram of body weight. An intake to permit satisfactory storage has been estimated at three times the minimum requirement. On this basis a baby at one year of age, weighing 10 kilograms, will have a minimum daily requirement of 200 units as vitamin A and 400 units as carotene; satisfactory intakes for storage would be 600 units as preformed vitamin A and 1200 units as carotene. On the basis of the belief that the requirement for the young is double that of the adult, a satisfactory intake would be 1200 units as vitamin A or 2400 units as carotene. Using the blood level as the criterion, Lewis and co-workers have placed the requirement for infants at between 100 and 200 units for each kilogram of body weight. An allowance of 1500 units daily for infants under one year of age has been recommended by the National Research Council; this allowance was intended to be an ample one.

A baby ingesting 100 ml. of human or cow's milk for each kilogram of body weight ($1\frac{1}{2}$ ounces to the pound) will receive from 100 to 200 units of total vitamin A for each kilogram of body weight from the milk alone. Such an amount should be adequate, especially since a considerable portion is present as preformed vitamin A. Results of studies with infants corroborate this view.

Observations have been made on the response in rate of growth and development of two groups of infants, one given only the vitamin A of the food, the other given a considerable addition. Such studies have shown no appreciable difference between the two groups. Similarly, studies of the incidence of infection in two such groups showed no significant difference, whether large amounts of vitamin A were given or only the vitamin A of the food.

Studies of dark adaptation in infants were initiated by Clemmesen of Denmark. The infant is held in the dark by a nurse until the child is quiet, but awake. A very dim light is moved across the child's field of vision several times. If the child turns his head toward the light, it is assumed that he has seen it; the least quantity of light that provokes this reaction is called the "minimum perceptible." Infants deficient in vitamin A need much more light than do normal infants. The test is thus similar to, but cruder than, those used for older children and adults. Studies carried out in this country, using this technique, indicate that the vitamin A content of the milk formula is ample for dark adaptation by the well infant. These studies, therefore, corroborate those in which the rates of growth and development were used as the criteria of adequacy.

It must be pointed out that illness, particularly infection, increases the requirement for or decreases the absorption of vitamin A markedly, and an amount of this vitamin that provides amply for the needs of the well child may be inadequate for the sick one. Infection greatly decreases

the absorption of carotene by infants, and the livers of infants dying after several days of illness often contain no vitamin A, in contrast to the relatively high vitamin A content of livers of infants after sudden death. When impairment of absorption of vitamin A exists, the amount in the diet should be considered insufficient, and an additional amount should be given, preferably as preformed vitamin A rather than as carotene.

Prematurely born infants at birth have a smaller store of vitamin A in the liver than do babies born at term. In addition, the prematurely born infant often absorbs fat poorly, so that the amount of fat-soluble vitamins, including vitamin A, absorbed may be inadequate. It is thus desirable to add vitamin A, as well as vitamin D, to the diet of the prematurely born infant soon after birth.

Hypervitaminosis A has been reported in a boy three years of age who had received daily about 340,000 units of vitamin A during most of his lifetime. The disturbance was characterized by hypoplastic anemia, leukopenia, increase in serum phosphatase, lipids and vitamin A, increased size of liver and spleen, abnormally rapid epiphyseal and carpal development together with decalcification of the skeleton. The child's hair was sparse and coarse and the fingers clubbed.

Vitamin D

Vitamin D is concerned with the utilization of calcium and phosphorus. Though rarely an infant may be encountered who has good utilization of these minerals with a vitamin D intake that would ordinarily be considered negligible, most infants in the temperate zone require vitamin D in addition to the minute amounts normally found in milk or in other food that the infant receives. When infants are deprived of vitamin D, the mineralization of bones and teeth is defective, and rickets develops. Infantile tetany often is related to vitamin D deficiency. Because vitamin

D is concerned with the mineralization and growth of the skeleton, it is related also to the growth of the body in length.

Vitamin D is present in milk, eggs, and a few other foods, but the amounts present are unimportant when considered in relation to the requirement of the growing infant and child. Ultraviolet rays from the sun, or from artificial sources, act on cholesterol in the skin, with the formation of vitamin D. The amount of vitamin D thus formed may be adequate for the baby's needs if the baby is sufficiently exposed. Such conditions may be attained by exposure to the sun during the summer, but rarely in the winter. It becomes necessary therefore, at least during the colder seasons, to supply vitamin D from special sources. In hot climates the baby usually is protected from direct exposure to the sun and may develop rickets as severe as is found in colder regions.

Several forms of vitamin D exist, but those most commonly in use are vitamin D₃, or activated 7-dehydrocholesterol, the chief form occurring naturally in cod-liver oil and vitamin D₂ (calciferol, activated ergosterol), commonly sold in this country under the name of viosterol. Apparently the two types of vitamin D are utilized differently by certain species, but it is probable that the difference in effect of the two forms on human infants is, at most, not marked. The effect of concentration of the vitamin on its utilization is probably much greater than the effect of the type of vitamin D. Dihydrotachysterol, called AT 10 (anti-tetany preparation No. 10), has some effectiveness as an antirachitic, but is definitely inferior to vitamins D₂ and D₃.

The question of the daily requirement of vitamin D by the well infant has been the subject of much discussion. Differences of opinion are largely due to the criteria used for determining the adequacy of dosage, and to the degree of control of the experimental work. It seems generally conceded that, for accurate studies, the work must be

carried on in winter and the infants must be inpatients under careful control twenty-four hours a day. Thus, it has been established that milk containing 135 U.S.P. or international units of vitamin D to the quart (140 units to the liter) will prevent the development of clinically significant rickets in the average full-term infant ingesting the quantities of milk usually fed. Such an infant receives from 60 to 135 units of vitamin D daily according to age, and the average intake for the first ten months approximates 110 units a day, a relatively small intake. It has also been established that these infants grow and develop at average rates.

Infants who are given 300 to 400 units of vitamin D daily as cod-liver oil, or who are fed milk with 400 units of vitamin D to the quart (415 units to the liter), permitting an average daily intake of about 325 units throughout infancy, retain more calcium and phosphorus and grow somewhat faster than infants fed 135-unit milk. Their average muscular development is more rapid, and dentition is earlier than in the preceding group. The question as to which rate of growth is to be considered "normal" may be answered by a study of infants fed the higher level but suffering from mild chronic infections during their first year. Such babies tend to grow and develop at a slower rate than their healthier mates and approximate the rate of those given the lower vitamin D dosage.

On the other hand, infants given 1800 to 2000 units of vitamin D daily as cod-liver oil, or as cod-liver-oil concentrate dispersed in milk, grow rapidly for the first few months, then slowly, so that the average rate of growth for infancy is far slower than that of the 300- to 400-unit group, and even slower than that of the babies given 135-unit milk. Decreasing the daily vitamin D intake to 400 units results in an increased growth rate within six to eight weeks. In a large outpatient study, infants fed 1500 units of vitamin D daily grew at the same rate as those fed 135-unit milk, a rate slower than that of infants given 300 to

400 units daily, but not so slow as that of infants given 2000 units or more each day. It appears from these studies that amounts of vitamin D much above 1000 units daily may be considered as moderately toxic for the infant.

A study of the efficiency of utilization of concentrated sources of vitamin D shows that infants vary widely in their ability to utilize the vitamin in concentrated form. A very few infants apparently utilize all the vitamin; many, judging from their retention of minerals and their rates of growth and development, are able to utilize only one-half or less of the total dosage. When concentrates are given in the amount of 4000 units daily, the infants show the same symptoms of excessive dosage as do those given 1800 to 2000 units as cod-liver oil or cod-liver-oil milk.

For these reasons it is concluded that the source of vitamin D fed to infants should preferably contain not more than 180 units to the gram of oil or other vehicle. An amount approximating 300 to 400 units daily is sufficient for sound growth and development and permits sufficient retention of calcium and phosphorus to provide a reserve supply in the bone for growth. It appears probable that the optimum vitamin D intake is close to this amount. A very low or very high vitamin D intake results in a slower and less steady rate of growth and development during the period of infancy.

It must be emphasized that the baby needs vitamin D additions as soon as possible. Cod-liver oil or other suitable form of vitamin D should be given in the first two weeks after birth and the dose should be increased as rapidly as possible until 300 to 400 units are ingested. If the use of concentrated preparations is considered desirable, allowance must be made for the lessened efficiency of utilization. Six hundred to eight hundred units daily should be ample.

Within recent years the use of a single massive dose of vitamin D has been advocated for prevention and cure of rickets. Doses as high as a million units of crystalline

vitamin D₂ (25 mg.) have been given to rachitic infants, and amounts of from 200,000 to 600,000 units (5 to 15 mg.) of vitamin D₂ and of vitamin D₃ have been administered for prophylaxis. The vitamin D has been given both orally and parenterally. Most of the reports of this procedure indicate adequate effectiveness in prevention or cure with no evidence of injury to the infants. However, the procedure must be considered as questionable until better evidence of lack of harm becomes available. If 2000 units daily for five to six months (total 300,000 to 360,000 units) cause slowing of growth, the effect on growth of from 200,000 to 600,000 units given in a single dose should be known before its use becomes common. The effects may be even more serious than slowing of growth. Some infants and children who have died after such therapy have shown calcification of various tissues and extreme renal damage. Some advocates of this type of therapy claim that the kidney damage, when present, must have preceded the vitamin therapy. However, it is known that one of the first evidences of hypervitaminosis D is increase of excretion of calcium in the urine, followed by increase in serum calcium. In an experiment in which 20,000 units of vitamin D for each kilogram of body weight was given to each of three dogs, the serum calcium of one dog remained above 12 mg. for each 100 ml. of serum for more than two months; the dog showed no gastrointestinal distress or other sign of toxicity.

Vitamin K

The name vitamin K (Koagulations-vitamin) was proposed by Dam of Copenhagen to designate a substance necessary to prevent a hemorrhagic disease of chickens. In this disease, which is characterized by low blood prothrombin values and a marked delay in the clotting of the blood, the addition of vitamin K to the diet increased blood prothrombin to the normal level. Vitamin K has since been found essential also for normal prothrombin

formation in man. It acts indirectly: that is, it is not a prosthetic group of prothrombin. Several hours are required for its effectiveness to be manifest, and presumably the liver is involved in the process. When the pure vitamin was isolated, it was shown to have a quinoid structure, and the substance was proved to be 2-methyl-3-phytyl-1,4-naphthoquinone. This substance has been given the non-proprietary designation "menadione." Many other derivatives of naphthoquinone have vitamin K activity, and some have the advantage of being water-soluble. The simplest of these is 2-methyl-1,4-naphthoquinone. The fat-soluble forms of the vitamin, commonly present in food, are not absorbed in the absence of bile salts. The water-soluble forms apparently are readily absorbed even in the absence of bile salts from the intestine.

Vitamin K adequacy in the infant may be measured by the amount of prothrombin in the blood. Several studies have shown that the prothrombin of newborn infants tends to be markedly lower than that of the maternal blood, and may be very low if the diet of the mother has been inadequate. A further lowering occurs between the first and sixth days. The levels of prothrombin may decline to as little as 10 per cent of the normal adult level, yet hemorrhage may not occur. In adults such a prothrombin level would be accompanied by serious hemorrhage. It thus seems clear that infants do not develop a tendency to bleeding unless the hypoprothrombinemia is extreme. Some factor or factors must operate in early infancy to increase utilization of prothrombin. The exact amounts of prothrombin reported in the blood of the newborn infant appear to depend, in part, on the method used. Values obtained with the two-stage test differ from those of the various one-stage tests. The rate of conversion of prothrombin to thrombin is influenced by several factors, some of which cannot be evaluated in the simpler tests.

Vitamin K given to normal newborn infants increases the amount of prothrombin in the blood. In normal infants

not given vitamin K, the prothrombin level begins to rise spontaneously on the sixth day after birth, after feeding has become established. It is known also that some intestinal bacteria can synthesize vitamin K. The prothrombin level of the blood does not reach adult level, however, until toward the end of the first year.

The blood prothrombin of prematurely born infants has been reported to be as low as one-third of that found in normal full-term infants, and only 10 per cent of the values of the maternal bloods. No hemorrhages were reported in the prematurely born infants studied.

Infants with hemorrhagic disease of the newborn show blood prothrombin values still lower than do normal infants. A rise in prothrombin occurs coincident with cessation of hemorrhage. When such infants are treated with vitamin K, hemorrhage ceases and prothrombin levels become equal to those of normal newborn infants. Hemorrhagic disease of the newborn is therefore considered to be the result of vitamin K deficiency. In hemolytic anemia of the newborn (the several varieties of erythroblastosis), the blood prothrombin content is greatly reduced, and may be nil. Evidence exists that the maternal supplies of vitamin K, along with vitamin C and certain minerals, are essential for early growth of the embryo. The two vitamins appear to be important in the prevention of abortion and of ante-partum bleeding.

Administration of vitamin K to the mother before parturition, or to the infant after birth, will prevent the usual decrease in prothrombin. Prophylactic administration of vitamin K to mothers at least four hours before parturition may be expected to eliminate hemorrhagic disease of the newborn and to reduce the incidence of intracranial hemorrhage. Vitamin K should be administered especially to mothers in cases of maternal toxemia, difficult or instrumental delivery, or premature labor. It should also be given to any newborn infant if cerebral symptoms of any

sort develop, as well as to all infants with hemorrhagic disease of the newborn, intracranial hemorrhage, icterus gravis, and anemia, and should be administered routinely whenever surgery is required during the first week of life. In cases of congenital obliteration of the bile ducts, a water-soluble vitamin K should be given, especially as a preliminary to surgical procedure. In prolonged vomiting the absorption of vitamin K will be poor, and hypoprothrombinemia may develop.

Infants can utilize at least several of the compounds with vitamin-K activity. If those soluble only in fat are used, absorption may be poor unless bile salts also are given. Those soluble in water are possibly preferable for infants.

Vitamin E

Vitamin E (alpha, beta, and gamma tocopherol) is essential for normal reproduction in animals. Its place in human nutrition has not yet been established. When deficient in the diet of the female animal, conception occurs, but the fertilized ovum is resorbed. When deficient in the diet of the male, spermatogenesis is defective. When minimum amounts are fed to the female, the young may be born alive, but suffer from muscle dysfunction and are weakly. Young suckling rats deprived of vitamin E develop a flaccid paralysis which can be cured by the ingestion of the vitamin. The muscle tissue presents the same appearance as the affected muscle of pseudohypertrophic muscular dystrophy of children. However, vitamin E therapy in muscular dystrophy has been wholly without success. Vitamin E has such a wide distribution in our foods that there would appear to be little opportunity for deficiency except as utilization may be impaired. Vitamin E acts as a valuable antioxidant, preserving other fat-carried vitamins in food from oxidation during storage, and protecting fat from development of rancidity.

The Vitamin-B Complex

Knowledge of the chemical nature and physiological effect of the vitamin-B complex has grown rapidly in the past few years. Originally known as "water-soluble B" and later separated into two components known as heat-labile and heat-stable fractions, it is now known to include many factors. Several components have been isolated and synthesized, and the presence of many others has been suspected. Those members most fully studied include thiamine (vitamin B₁, aneurin), riboflavin (vitamin B₂, vitamin G) and nicotinic acid (niacin) and its amide. Pyridoxine, pantothenic acid, biotin, folic acid, choline, inositol, para-aminobenzoic acid, and probably several other vitamins are also included in the complex. Some of these appear to be essential for certain species only; certain others determined but vaguely may turn out to be merely definite proportions of the vitamins already isolated.

In general, it may be said that the members of the vitamin-B complex tend to occur together in nature, though the relative proportions of the different members in any one food may differ. For example, cow's milk contains more riboflavin in proportion to the other B vitamins than is found in meat or wheat germ. The proportions of the different members of the B group differ in wheat germ, yeast and liver, yet all are considered excellent sources of the complex.

Physiologically the action of the group is somewhat similar. Thiamine, riboflavin, nicotinic acid, and probably other members of the B complex, each forms a part of a complex enzyme molecule belonging to the group of so-called respiratory enzymes and associated in the body with oxidation-reduction (respiratory) reactions of the cell. Each is combined with phosphate, a protein, and usually some other molecule to form the enzyme. The "yellow enzyme of Warburg" is composed of protein, riboflavin, pentose, and phosphate, and activates molecular oxygen so

the cell can use it. The enzyme carboxylase is made up of protein and thiamine pyrophosphate. The respiratory enzyme of Warburg is made up of a protein, adenine, two pentose molecules, three phosphoric acid molecules, and nicotinic acid amide.

Obviously a deficiency of a single B vitamin rarely if ever occurs. In fact, it is now usually considered that no human vitamin deficiency is single; all are multiple, though the lack of one vitamin may predominate. In any multiple deficiency disease the giving of a single vitamin may result in the alleviation of one set of symptoms, but usually with exacerbation of other symptoms previously unnoted or considered minor. For this reason it seems wiser for prophylactic purposes to obtain vitamins from food than from concentrates, and in any deficiency ample intake of all known vitamins should be provided. Particularly must such patients be watched for manifestations of other vitamin deficiencies.

Thiamine

Thiamine (vitamin B, vitamin B₁, aneurin, antiberiberi vitamin, antineuritic vitamin) is a water-soluble compound containing both nitrogen and sulfur, and is related to the pigment thiochrome. It is easily destroyed by heat. The usual synthetic product in commerce is the hydrochloride of thiamine.

Plants synthesize thiamine. It is stored in the bran coats of seeds, and to a lesser extent is formed in leaves. Thiamine is contained in animal tissues, milk and eggs. Organ meats contain the highest concentration. It is not stored in the animal body to any great extent. Yeast cells and some bacteria, including certain intestinal flora, synthesize thiamine.

The occurrence of thiamine deficiency in man proves that the intestinal flora cannot be depended on to produce sufficient thiamine to meet human requirements. It is possible that certain precursors must be provided. Synthesis of varying amounts of thiamine in the intestine is a prob-

able cause for the discrepancy in apparent thiamine requirement noted by different observers.

Thiamine occurs in the body both free and as the pyrophosphate ester and is a component of one or more of the respiratory enzymes concerned in the utilization of carbohydrate by the tissues. The enzyme carboxylase is composed of a protein, a metallic ion, probably magnesium, and thiamine pyrophosphate. The nonprotein part of an enzyme is called a coenzyme. Thus, thiamine pyrophosphate is spoken of as cocarboxylase. In the absence of carboxylase, carbohydrate oxidation is incomplete and as a result pyruvic acid collects in the tissues and blood. Thiamine alone is ineffective in oxidizing pyruvic acid, but thiamine pyrophosphate (cocarboxylase) is very effective. It is thought that all nucleated cells can phosphorylate thiamine, so that the vitamin may be ingested either as thiamine or as cocarboxylase and be efficiently utilized.

Formerly vitamin B₁ was assayed and spoken of quantitatively in terms of units. Since the isolation of thiamine this material is referred to in terms of weight. One microgram or one gamma is one one-thousandth of a milligram and is the equivalent of one-third of a unit.

The daily requirement of thiamine depends on the amount of carbohydrate to be oxidized. Apparently thiamine-containing enzymes are not needed in the breakdown of fat by the body, so the requirement is related directly to the nonfat calories of the diet. Thus a baby whose energy requirement is satisfied by a low-fat diet would have a higher requirement of thiamine than a baby whose energy need is supplied by a diet made up largely of whole milk.

No close agreement yet exists as to the daily requirement of the baby for thiamine. The League of Nations standard (1935) was 150 micrograms (50 units) daily. The minimum requirement adopted by the U. S. Food and Drug Administration for regulatory purposes is 225 micrograms as an average for the first year of life. The Food and Nutrition Board of the National Research Council has

stated 400 micrograms as a recommended allowance (not minimum requirement) at the age of six months.

Another method of statement that has had the sanction of widespread usage is that the minimum requirement is 30 micrograms for each 100 calories of food ingested. On this basis the recommended allowance should be 50 micrograms for each 100 calories.

The thiamine content of human milk depends on the amount ingested by the mother. It is difficult to increase the thiamine content to more than 27 to 33 micrograms to each 100 ml. (8 to 10 to the ounce) and the average is much less than this amount, in the neighborhood of 15 micrograms to each 100 ml. (3 to 5 micrograms to the ounce), after lactation is established. During the first two weeks of lactation the thiamine content of human milk varies from 0 to 9 micrograms to each 100 ml.

Cow's milk usually contains more thiamine than does human milk, but the amounts are widely variable. Various analyses of cow's milk place the content of thiamine at from 17 to 70 micrograms to each 100 ml. (5 to 22 to the ounce), with the average value about 38 micrograms to each 100 ml. (12 to the ounce). During pasteurization approximately 10 per cent is lost. Milk that is boiled for a short time (three minutes) loses approximately 10 per cent of its thiamine. During commercial evaporation the loss is approximately 20 per cent. Storage of evaporated milk at room temperature causes a continued loss of thiamine, producing a total loss after eight months' storage of amounts up to 40 per cent of the quantity present in the raw milk from which it was prepared.

The components of the baby's diet other than milk contain small amounts of thiamine. As fed, orange juice, tomato juice, sieved peas, and whole grain cereals provide 60 to 80 micrograms for each 100 ml. (18 to 25 to the ounce). An egg yolk provides 50 to 75 micrograms. Sieved fruits vary in thiamine content from 0 to 65 micrograms to each 100 ml. (0 to 20 to the ounce).

If the requirement for thiamine of 30 micrograms for each 100 calories is acceptable as a minimum standard, human milk should contain 20 micrograms to each 100 ml. (6 to the ounce) in order to carry its own share. Such a level can be achieved only when the maternal diet contains ample amounts. Much evidence exists that the diet of many women contains suboptimum amounts of thiamine. Clements has reported the occurrence of thiamine deficiency among breast-fed babies in Australia. The milk ingested by these babies contained less thiamine for each 100 ml. than the milk of mothers whose infants were thriving. All breast-fed infants presumably ingest very little thiamine during the first two weeks after birth. Such stores as were present at birth are probably depleted within a few days.

Mild thiamine deficiency in infants is associated with symptoms not specific for the disturbance. Failure to gain properly, constipation, vomiting, restlessness, and fretfulness are reported. All symptoms are alleviated within a few days by administration of thiamine. As the deficiency becomes more severe, the infants become flabby and emaciated, and the facial expression is that of suffering. These infants are very susceptible to infection. In still more severe thiamine deficiency edema is commonly present. Thiamine deficiency in pregnant women is associated with retarded growth or death of the fetus.

The artificially fed baby fares somewhat better as regards vitamin B₁, when customary formulas are prepared from boiled fresh milk of average thiamine content. Such formulas contain their share of the requirement. Formulas prepared from evaporated milk, however, do not contain quite their share of the requirement unless the product is less than four months old. The average lapse of time between production of evaporated milk and its retail sale has been estimated to be three and one-half months. The customary addition of an ounce of orange juice to the diet in

the early weeks of life usually may be considered as adding enough thiamine to meet minimum requirements, even when boiled or evaporated milk of low thiamine content is used. Often, however, the thiamine intake is not optimum until additional dietary supplements are given.

Riboflavin

Riboflavin (vitamin B₂, vitamin G, lactoflavin) has been separated in pure form and also synthesized. It is a water-soluble, deep yellow pigment, with a marked green fluorescence. In its pure form it is sensitive to light, but in foods it appears to be somewhat protected. The riboflavin content of milk is decreased rapidly on exposure to sunlight. Riboflavin is a constituent of several known enzymes, either as riboflavin phosphate (riboflavin mononucleotide) or as riboflavin adenine dinucleotide.

Riboflavin is essential for human beings. The most striking evidences of deficiency are changes in the eyes and about the mouth. One of the earliest changes is an injection of the conjunctiva about the cornea. Associated with this change are photophobia, a burning sensation of the eyeballs and dimness of vision referred to as "twilight blindness." As the eye lesion progresses, the cornea becomes vascularized and interstitial keratitis results. When the mouth is affected, a specific type of glossitis may be present; the more characteristic lesion is a chronic inflammation of the lips (cheilitis), especially at the angles of the mouth. Seborrhea of the nasolabial folds, *ali nasi*, eyelids and ears also occurs. All these lesions are cured by the administration of riboflavin when caused by riboflavin deficiency. Some of them may be caused by other conditions.

Acute riboflavin deficiency has been produced in dogs, resulting in sudden collapse with marked fall in body temperature and respiration rate. The collapse is supposedly due to deficient oxidation of carbohydrate, espe-

cially by the brain, due to the lack of the respiratory enzyme of which riboflavin is a part. In chicks and in pigs deficiency of riboflavin may result in irreparable damage to main nerve trunks. Riboflavin deficiency in pregnant rats results in congenital deformities of the eyes and the skeleton in the young. Riboflavin deficiency has not been proved a cause of congenital malformation in the human infant.

The quantity of riboflavin in human milk varies with the intake of the mother. Reported values vary from 15 to 50 micrograms for each 100 ml. (4 to 14 micrograms to the ounce), with an average value of 40 micrograms for each 100 ml. in this country. Cow's milk contains more than three times as much riboflavin as human milk, 180 to 200 micrograms for each 100 ml. or 55 to 60 micrograms to the ounce. The riboflavin content of milk is apparently unaffected by heat treatment. Other good sources of riboflavin for the infant are liver, egg yolk, and green vegetables, which add 750, 1600 and 15 to 70 micrograms, respectively, for each ounce (30 grams) fed. The question of synthesis of riboflavin by intestinal flora, particularly of breast-fed infants, needs further study.

Animal experiments indicate that the riboflavin requirement is approximately 50 per cent greater than that for thiamine. The minimum daily requirement for the infant is given by the U. S. Food and Drug Administration as 500 micrograms (0.5 mg.). The recommended allowance of the National Research Council is 600 micrograms (0.6 mg.) for the midportion of the first year. The infant fed customary formulas of cow's milk has an intake well above this minimum. The breast-fed baby must ingest a liter or more of milk daily to obtain the minimum amount stated. If the mother's diet is poor in riboflavin, the intake of the baby may be well below the minimum. The early addition of other foods to the diet seems desirable, particularly when the mother's diet is poor.

Nicotinic Acid

Nicotinic acid or its amide (Niacin, P-P or pellagra preventive factor) is the chief food component required for the prevention of the pellagra syndrome. The compound has been known as a chemical entity for years, but only recently has it been proved to be the chief effective agent in pellagra prevention. A diet deficient only in nicotinic acid is practically impossible to obtain, except under intentional and experimental conditions. Consequently, pellagra is nearly always associated with symptoms of deficiency of several of the B-complex factors.

Nicotinic acid, like other members of the vitamin-B complex, is a component of oxidation-reduction (respiratory) enzymes of the body. The compound has been synthesized and it has been shown that related compounds convertible to nicotinic acid by the body are equivalent to the pure nicotinic acid itself. It occurs in the enzymes probably in the form of its amide.

The metabolism of nicotinic acid seems to be linked with that of iron. Dogs with nicotinic acid deficiency develop severe macrocytic anemia which responds to nicotinic acid therapy. The conspicuous pigment of pellagrins contains iron. Porphyrinuria is a relatively early sign of nicotinic acid deficiency. Some evidence that nicotinic acid affects the iron containing enzyme systems has been reported.

The requirement of the infant for nicotinic acid cannot be stated at present with accuracy. The adult requires approximately 15 mg. daily for prevention of pellagra, and 20 mg. is commonly stated as a satisfactory intake. If the requirement is proportional to the energy intake, the infant would require one-third or less of the amount necessary for the adult. Animal experiments indicate that the requirement for nicotinic acid is 8 to 10 times that for thiamine at all stages of development.

The nicotinic acid content of cow's milk is approximately 1 mg. to the liter or 30 micrograms to the ounce. Human

milk contains about 1.8 mg. to the liter. An egg yolk contains about 200 micrograms and beef liver 14.2 mg. for each 100 grams. Tomato is a poor source of nicotinic acid. Potato and spinach provide 1.5 to 2.0 mg. for each 100 grams (450 to 600 micrograms to the ounce). Milk is a poor source of nicotinic acid compared with the other foods reported. Its high content of some of the other factors of the vitamin B complex may account in part for its efficacy in the treatment of pellagra. It is possible also that milk may contain substances convertible to nicotinic acid within the body. Such substances have been found in wheat bran. Some evidence exists also that nicotinic acid may be synthesized by bacteria in the intestinal tract. The conditions favorable to such synthesis have not been determined. The efficacy of milk in prevention and treatment of pellagra is related also to the finding that nicotinic acid and tryptophane are complementary to each other. The addition of tryptophane to a diet will compensate for moderate deficiency of nicotinic acid. It is noteworthy that corn, a prominent component of the diet in pellagrous regions, has a relatively low content of both nicotinic acid and tryptophane and also probably contains an "antivitamin" or structural analogue of nicotinic acid which competes with the vitamin in the body but has no vitamin effect. Tryptophane and nicotinic acid probably both counteract the anti-vitamin.

Nicotinic acid deficiency is reported to cause cyanotic attacks, spasticity and convulsions in infants. Pellagra is a multiple deficiency disease involving nicotinic acid and other B-group vitamins. It occurs in infants whose mothers have had inadequate diets during pregnancy and lactation. Such women secrete milk probably of poor quality, and often of insufficient quantity, so that the infant must be weaned early. The diets after weaning are usually inadequate. The infants develop poor appetites and become irritable and apprehensive, with restless sleep. Constipation is common but may alternate with periods of diarrhea.

Soreness of lips and tongue is a frequent symptom, but the typical lesions of pellagra are rarely seen in infancy. Response to nicotinic acid therapy is dramatic. It is suggested that in areas where pellagra is endemic, the presence or absence of pellagra should be tested by means of the response to nicotinic acid. It has been reported also that Vincent's angina is relieved by the administration of nicotinic acid. The rationale of this procedure is to achieve normality of the mucous membrane of the mouth and throat, so that it is able to resist bacterial invasion.

Folic Acid

Folic acid (pteroylglutamic acid, L. casei factor, vitamin B_c, vitamin M) is a constituent of the vitamin B complex, the isolation and identification of which have been approached through various channels by several observers. Because of the diversity of approach, it is not yet wholly clear whether all the products included under this designation are identical. A factor has been isolated from liver, from yeast, and from leafy vegetables that is necessary for hematopoiesis of various species, including man. It has been found to be necessary also for the growth of certain bacteria, notably *Lactobacillus casei* and *Streptococcus lactis* R. Early it was designated as the norit eluate factor. Subsequently this factor or a similar one was called vitamin B_c, the c representing the relationship to the chicken, in which species the factor prevents anemia. The term folic acid was derived from folium (leaf) and it is now used synonymously with the term L. casei factor. Folic acid (L. casei factor) is now commercially available in pure crystalline form and the factor represented by this crystalline form is the one commonly referred to as folic acid.

Folic acid has been shown to be effective in producing recovery from certain macrocytic hyperchromic anemias in man. It has been found to be effective in the treatment of the anemia of sprue and to some extent in the anemia of

celiac disease. Used alone, it does not give complete relief in pernicious anemia, especially from the neurological symptoms.

Pyridoxine

Pyridoxine (vitamin B₆, adermin, rat antidermatitis vitamin, rat acrodynia vitamin) is widely distributed in plant and animal tissues. The germ and bran coats of seeds are good sources. Organ meats contain moderate amounts. The content of human milk is approximately the same as that of cow's milk, about 65 micrograms for each 100 ml.

The effect of pyridoxine deficiency has been studied largely in animals; acrodynia-like dermatitis, epileptiform convulsions and disturbances of the hematopoietic system have been noted. Dogs and pigs with pyridoxine deficiency develop microcytic hypochromic anemia needing both pyridoxine and iron for cure. Evidence exists that pyridoxine may be concerned with utilization of unsaturated fatty acids; with conversion of protein to fat; with regulation of tryptophane metabolism; with a coenzyme for certain transaminases. It also appears that pyridoxine may be of importance in maintenance of normal lymphoid tissue and that deficiency produces suppression of antibodies.

The human requirements for pyroxidine are unknown. It must be assumed that the needs of the infant are supplied by customary diets.

Biotin

Biotin (vitamin H, factor W) is classed with the B group of vitamins. It is widely distributed in plant tissues and occurs also in milk, eggs, and especially in liver. Deficiency in animals results in seborrheic desquamation of the skin. Experimental deficiency in man has been produced by feeding large amounts of raw (dried) egg white, which contains a substance avidin, capable of destroying biotin. Such deficiency results in a striking ashy pallor of the skin and mucous membranes, followed by dryness and

desquamation. Lassitude, anorexia, muscle pains, and precordial distress were noted. All symptoms were relieved by administration of biotin. Deep brown pigmentation of the back has been reported in infants as a sign of biotin deficiency.

Choline

Choline has been known for many years as a part of the lecithin molecule. Its role in physiological processes has been elucidated only recently. It appears to have at least three functions in the body. (1) It is necessary for the mobilization of fat through the formation of phospholipid. In the absence of choline, fat accumulates in the liver rapidly. Deficiency of other of the B vitamins, notably thiamine and pyridoxine, is also associated with the production of fatty liver. (2) Choline is a constituent of acetyl choline, so necessary in the transmission of nerve impulses. (3) Choline is a source of labile methyl groups. Choline can be synthesized in the body to a certain extent from other compounds, as methionine and betaine, which contain methyl groups. The amount so formed, however, is inadequate.

Choline given to dogs decreases the rate of formation of red blood cells.

Other B Complex Factors

Members of the vitamin B complex other than those discussed in the foregoing include pantothenic acid, inositol, and para-aminobenzoic acid. Little is known of the requirement of these vitamins by the human. Studies with animals indicate that pantothenic acid is necessary for growth, and in at least one species its deficiency is associated with hemorrhage in the adrenal glands. Inositol is said to be concerned in the regulation of gastrointestinal peristalsis. It increases the mobility of the tract and nicotinic acid supposedly decreases mobility. Inositol is stated also to be lipotropic. Para-aminobenzoic acid inhibits the

action of the sulfonamide drugs, due to a competitive effect between the two compounds. It is said to be effective as a detoxicant for arsenicals.

It is specific in the treatment of rickettsial diseases; its action is said to be due to increasing the activity of cellular enzymes.

Ascorbic Acid

Ascorbic acid (vitamin C) is a hexose acid essential to man, other primates, and the guinea pig. Other animals studied have the ability to synthesize it. It has been synthesized in the laboratory and on a commercial scale. Ascorbic acid is a strong reducing agent and therefore is easily oxidized with loss of its vitamin activity. Destruction is favored by the presence of oxygen, heat, and light. Copper, even in minute amounts, acts as a catalyst in rapid destruction. Destruction is more rapid in alkaline and neutral media than in an acid medium.

The exact function of vitamin C in the body is not yet fully understood. A part of the functions ascribed to ascorbic acid may be due to vitamin P; the situation needs clarification. The theory that it acts as a respiratory catalyst is not well supported. It plays an essential role in growth processes and is found most abundantly in actively growing tissues. Its relationship to enzymes is not yet clear. Experimental evidence indicates that at least for certain poisons ascorbic acid is a detoxifying agent. The effects of deficiency are noted chiefly in the bones, teeth, and blood vessels, but probably this material is necessary for all living cells. Scurvy is the clinical entity produced by marked deficiency. In scurvy one of the most striking clinical changes is produced by hemorrhages caused by seepage of blood through capillary walls. The changes in the capillary walls seem to be dependent primarily on growth failure of the connecting-tissue supporting structure, with perhaps loss of cement substance between cells. Vitamin C is important for maintaining the normal status of connective-tissue cells and other similar

cells, also of the bones and teeth. The pathological changes are discussed further under Scurvy.

Ascorbic acid occurs in many fruits and vegetables. Citrus fruits, strawberries and some other berries, pineapple and tomato are good fruit sources, especially the first two groups. Vegetables of the cabbage, pepper, and turnip families, green leafy vegetables as cress, parsley and the familiar garden greens are excellent sources. The "pot-liquor" or water in which the foregoing vegetables are cooked is a cheap source for the infant. The vitamin-C content of cow's milk depends on precursors in the feed; the quantity of the vitamin is greater in spring and summer milk. Milk fresh from the cow may normally be expected to contain 20 mg. to the liter, or even slightly more. Pasteurization decreases the content approximately one-third. Further decreases occur during the time elapsing between milking and retail distribution. Exposed copper in the pasteurizer or cooler causes almost complete loss. Thus, market milk in a city will have a much lower content of vitamin C than that of the original milk. Values as low as 2 mg. to the liter have been found for commercially pasteurized milk three days old. Even though well-handled milk should contain an amount of vitamin C that will supply the baby with an important part of his requirement, this source of the vitamin is not sufficiently dependable. For this reason it has become customary in this country to consider the infant's milk formula as possibly devoid of vitamin C and to supply the daily need from supplements.

Though evaporated milk usually is processed fairly promptly after milking, the greater heat treatment causes greater losses of vitamin C than in the case of pasteurization. An average vitamin-C value for reconstituted evaporated milk is approximately 6 mg. to the liter. Possibly because of the shorter period of heat treatment, reconstituted dried milk has its vitamin-C content reduced to a lesser degree, with an average value of approximately 12 mg. to the liter.

The amount of ascorbic acid in human milk varies with the amount of vitamin C in the food. If the mother's intake is large, the milk may contain from 60 to 75 mg. to the liter. The average value reported in this country is approximately 50 mg. to the liter. If the mother's vitamin-C intake is low, the amount in the milk may decrease to practically zero.

Of the supplementary foods customarily given infants, the citrus fruits have the highest content of vitamin C. Orange juice, lemon juice, and grapefruit juice contain an average of 0.5 mg. to each ml., or 15 mg. to the fluid ounce. Tomato juice contains from one-third to one-half as much as orange juice. Canned orange juice and tomato juice are good sources. Cabbage juice and "pot liquor," or juice of boiled greens, are good sources of the vitamin which have the advantage of being cheap. Extracts of rose hips and of growing tips of some evergreens have been used in Europe as good sources of vitamin C. Cooked, sieved fruits ready for feeding contain from 0.5 to 2 mg. to the ounce (30 grams), sieved vegetables 0.5 to 6 mg.

An intake of 10 mg. of ascorbic acid daily is sufficient to prevent scurvy in the well infant, but not sufficient to maintain a satisfactory blood level or tissue concentration. One of the criteria that has been used in an attempt to determine the vitamin-C requirement is the amount of this material in the blood. All animals that synthesize their own vitamin C have approximately the same blood level, viz., 1.2 mg. to each 100 ml. Certain observers believe that an amount of ingested vitamin C that will maintain this blood level should be regarded as the optimum requirement. For the infant, such an intake would be 75 mg. or more daily, an amount that rarely is received by the baby from foods, though it is an amount that could be received from human milk if the mother's diet contained an abundance. Ascorbic acid in the amount of 20 mg. daily, given to the artificially fed young infant in addition to whatever may be in the milk formula, has been found to permit blood levels

approximating those of babies receiving human milk of average vitamin-C content. For older infants, 50 mg. daily may be required to permit this same blood level. Therefore, the daily requirement of the infant is commonly accepted as 20 to 50 mg. daily, depending on the age.

The concentration of ascorbic acid in the blood of the newborn is greater than that in the blood of the mother, and is usually above 0.7 mg. and often above 1 mg. to each 100 ml. The amount decreases rapidly in the artificially fed baby so that by the tenth day the average amount is less than 0.4 mg. to each 100 ml., or at the prescorbutic level. Unless vitamin C is added to the diet in sufficient quantity, the vitamin C of the blood remains very low. The breast-fed baby whose mother is ingesting ample amounts of vitamin C shows a fairly rapid increase in the vitamin-C content of the blood soon after the milk flow is established. The baby who is fed heat-treated cow's milk, or human milk from a woman whose intake of vitamin C is low, cannot maintain a satisfactory blood ascorbic acid level: vitamin C must be given. As the blood level is low by the tenth day, it seems highly desirable to add a vitamin-C supplement at that time, particularly for artificially fed babies, instead of waiting until the child is three or four weeks of age. Beginning with one teaspoonful of orange juice, the healthy baby will soon tolerate one to one and one-half ounces (30 to 45 ml.) daily. This amount seems sufficient for the very young infant, but after three months of age two to three ounces (60 to 90 ml.) a day are desirable, or an equal amount of the vitamin from other sources. For the older baby, foods other than orange juice contribute materially to the vitamin-C intake, but these must be chosen with care and intelligence if the stated requirement of 50 mg. is to be met.

Claim has been made that the breast-fed baby does not need orange juice or other source of vitamin C. No doubt such a claim would be correct if the mother's intake of vitamin C were ample. In existing circumstances the require-

ment of the infant may not be fully met from this source alone. The addition of orange juice to the diet of the baby makes it more certain that he will receive an ample supply.

It has been shown that the vitamin-C requirement is increased by infection. The present-day practice of giving additional fruit juice to the sick infant or child is thus sound from the standpoint of vitamin-C intake, as well as of increased fluid intake.

Vitamin P

Evidence has accumulated concerning the existence of a water-soluble vitamin, called vitamin P, which is present in citrus fruits and which is the factor responsible for the integrity of the capillary wall. Citrus fruits seem to be the richest source of this vitamin so far investigated. Other good sources are grapes, plums, and prunes. The activity of the vitamin appears to diminish with storage of the fruit.

Szent-Györgi, who first discovered vitamin P, isolated from Hungarian red pepper and from lemon juice a flavone glucoside, called citrin, which was active in treatment of increased permeability of capillaries. Later, Szent-Györgi separated citrin into two components, the glucoside hesperidin and the glycoside of eriodictyol. Others believe that the eriodictyol is the chalcone of hesperidin. Another flavone glucoside, rutin, which is structurally related to hesperidin, is also stated to have vitamin P activity.

The relation of vitamin P to the requirement of ascorbic acid probably needs reinvestigation. The finding by Meyer and Hathaway that orange juice is more effective for small children than its ascorbic acid equivalent plus potassium citrate may have some bearing on this question.

CHAPTER VIII

SUMMARY OF THE NUTRITIONAL REQUIREMENTS OF INFANTS

For ready reference, the nutritional requirements of the infant as detailed in the preceding six chapters are here briefly summarized.

Calories

During the first year of life the normal infant should receive daily an average of 50 to 55 calories for each pound of body weight (110 to 120 calories for each kilogram). The caloric requirement is somewhat greater than the above during the first three months of life, and somewhat less after the sixth month. The caloric requirement of the undernourished or overnourished infant should be calculated on the basis of the *expected* or normal weight for the age. In the case of the normal breast-fed infant, the caloric requirement is met if the infant receives daily approximately 2.5 to 3 ounces of milk for each pound of body weight (165 to 200 ml. for each kilogram). In the case of the young artificially fed infant, approximately two-thirds of the total caloric requirement should be met by milk and one-third by added carbohydrate. Toward the end of the first year other foods of the diet replace the sugar added to the milk.

Proteins

The protein requirements of the normal infant are adequately met when he receives daily approximately 2.5 ounces of human milk for each pound of body weight (165 ml. for each kilogram), or 1.5 to 2 ounces of cow's milk for each pound of body weight (100 ml. to 130 ml. for each kilogram). These amounts correspond to approximately 2 grams of protein for each kilogram in the case of the

breast-fed infant, and 3.5 grams for each kilogram in the case of the artificially fed infant. A moderate excess of protein does no harm. Undernourished infants should receive an amount of protein proportionate to the expected or normal rather than the actual body weight.

Carbohydrates

An infant should receive daily not less than 1 per cent of his body weight in carbohydrate (0.15 ounce for each pound or 10 grams for each kilogram). The breast-fed infant receives this amount in the milk. In the case of the artificially fed infant, approximately one-third of the carbohydrate should be derived from the milk and the remainder should be added in the form of sugar or starch. For most infants the proportion of carbohydrate added to cow's milk in the diet should be approximately one part of carbohydrate for each 10 to 15 parts of milk. Carbohydrates of the dextrin and maltose types are extensively employed during early infancy. However, sucrose or lactose may be used. After the fifth or sixth month, a portion of the carbohydrate should be given in the form of starch.

Fats

No definite amount of fat is necessary in the diet of the infant. Fats, however, are valuable sources of energy and unless they are included in the diet, large amounts of protein and carbohydrate are needed. When breast-fed or artificially fed infants are given the amounts of milk necessary to supply protein, a suitable amount of fat is also given. Milk from Jersey or Guernsey cows, containing a high percentage of fat, should not ordinarily be used for the feeding of infants, nor should top milk or cream be used for the preparation of the usual formulas. Skimmed or partially skimmed milk is often indicated in the presence of gastrointestinal disorders and for prematurely born and other weak infants whose utilization of fat is poor.

Mineral Salts

The breast-fed infant, receiving daily 165 ml. of milk for each kilogram of body weight (2.5 ounces to the pound), or the artificially fed infant receiving 100 to 130 ml. of cow's milk for each kilogram of body weight (1.5 to 2 ounces for each pound), receives sufficient mineral constituents with the exception of iron and possibly iodine. Iron-containing foods or a small amount of iron should be added to the diet of all infants after the third month of age, earlier in the case of those who are anemic. Iodine additions are indicated only in regions where goiter is prevalent.

Water

The infant's requirement for water is variable and depends on activity, environmental temperature, and the presence of diarrhea or vomiting. The daily requirement for the normal infant varies from 10 to 15 per cent of the actual body weight (1.5 to 2.5 ounces for each pound). Breast-fed infants usually receive sufficient water in the milk. Artificially fed infants, given undiluted whole milk formulas, should be offered additional water between feedings, especially in warm weather.

Vitamins

Vitamin D should be added to the diet of all infants, beginning as soon as feeding is established. The oily sources of vitamin D best utilized by infants are those containing less than 200 units to the gram. Thus the unconcentrated fish-liver oils are preferable to more concentrated oily sources. Sources of vitamin D soluble in water, or emulsifying easily with water, may be given in the feeding. Oily sources should always be fed separately in an amount providing 300 to 400 units daily. If oily concentrates are fed, the unitage should be increased by 50 per cent. The vitamin-D milks providing 400 units to the quart (415 to the liter) provide sufficient vitamin D for the healthy infant who ingests 100 ml. or more for each kilogram (1.5 ounces for each pound) of body weight or a total

of 720 ml. (24 ounces) daily. The use of single massive doses of vitamin D is not advised.

The amount of vitamin A in customary cow's milk formulas, together with the amount ingested by an infant receiving the usual supplements, is probably ample for the well infant. The amount of vitamin A in human milk seems to be approximately the same as that in cow's milk. Infants whose vitamin D is supplied by fish-liver oils get an additional amount, making the daily intake adequate under all circumstances except as illness affects utilization adversely.

An addition of vitamin K to the diet seems desirable during the newborn period. The need for additional amounts after this period is not established. The need for vitamin E has not been established for infants.

Vitamin C, like vitamin D, is needed from earliest infancy. Human milk contains an adequate amount if the diet of the mother contains an ample supply of the vitamin. For the artificially fed baby and for the breast-fed baby whose mother's diet is poor, orange juice should be given at or before two weeks of age, increasing the amount until 30 ml. (one ounce) is taken daily. After three months of age the daily intake should be increased to 60 ml. (two ounces). If crystalline ascorbic acid is given, a desirable intake is 20 mg. for the younger infant and 50 mg. daily for the infant over six months. Natural sources are preferable to synthetic because of possible need of the substance designated as vitamin P.

Of the B group of vitamins, it seems certain that no additional riboflavin is needed by the infant fed appropriate amounts of cow's milk. It appears probable also that at least the minimum requirement of the other members of the group is met by modern standards of infant feeding. The intake of the breast-fed infant depends so largely on the amount of these vitamins received by the mother that the danger of insufficiency of these vitamins may be very real in the low-income groups in which subacute or acute vitamin deficiency is prevalent among adults.

CHAPTER IX

DIGESTION IN INFANCY

The food of the infant is of necessity simple, consisting chiefly of milk and carbohydrate. The infant's digestive organs are well adapted to care for human milk and for somewhat lesser amounts of cow's milk. In proportion to his weight, however, the infant can digest a very large amount of milk and sugar. If an adult were to take as much cow's milk in proportion to his weight as a normal infant, he would be taking 8 to 10 liters a day, and would in addition be eating from 450 to 700 grams of sugar. However, the infant's capacity for the digestion of other foods is distinctly limited and is developed only as the infant grows older.

On account of the proportionately greater food requirements of the infant, the digestive functions are taxed closer to the limits of capacity than in the case of the adult, and there is a smaller margin of safety. Furthermore, the digestive functions of the infant are especially likely to become impaired as the result of such conditions as malnutrition, infection, fever, high external temperatures, or pain. Not infrequently the infant's digestive capacity is lowered to such an extent that the optimum amounts of food required for nutrition are in excess of his digestive capacity.

Salivary Digestion

Salivary digestion is relatively unimportant during early infancy, inasmuch as very little saliva is secreted during the first few months, and furthermore, saliva has no effect upon the constituents of milk. Later, when starch is fed, slight digestion may be accomplished by the enzyme ptyalin, and such digestion continues for a short period after the

food reaches the stomach. Salivary digestion assumes relatively little importance until the time the infant begins to chew starchy foods, such as toast, crackers, or cereals.

Gastric Digestion

The gastric glands are functionally active at the time of birth and capable of secreting hydrochloric acid, pepsin and rennin. The concentration of acid secreted by the infant is much less than that of the adult. There is a gradual increase in the strength of acid secreted throughout infancy and childhood. Considerable individual variation exists, some very young infants having a fairly concentrated gastric juice.

The total volume of the gastric secretion, as well as its acid content, is influenced by a variety of factors. The amount and concentration are greatly decreased in the presence of infections accompanied by fever. They are decreased in excessively hot weather and also as the result of pain or fright. The gastric secretion of undernourished infants is, in most instances, less than that of normal infants of the same age. As the result of pharyngeal, esophageal or gastric irritation, much alkaline mucus is secreted and this, when mixed with the gastric contents, partially neutralizes the acid and renders the gastric juice relatively inefficient. Gastric secretion is increased by the feeding of certain types of food, notably those with a high protein content. Some of the amino acids, especially beta-alanine, have a marked stimulating effect on gastric secretion. Commercial beef extract contains a considerable amount of beta-alanine and other substances capable of stimulating gastric secretion. Acidified foods have a similar effect. Psychic factors also play an important role in influencing gastric secretion.

Of the constituents of the gastric juice, hydrochloric acid, pepsin and rennin, the acid appears to perform the most important functions during infancy. In the case of the

normal breast-fed infant, the amount of hydrochloric acid secreted is sufficient to render the stomach contents distinctly acid at the height of digestion. The average degree of acidity attained corresponds to a hydrogen-ion concentration of pH 3.6. This degree of acidity is sufficient to inhibit markedly the growth of the majority of the bacteria likely to be introduced into the stomach. The acidity of the gastric contents is also one of the factors in regulating the pyloric reflex. On the passage of the stomach contents into the duodenum, the acid present under normal conditions is sufficient when coming in contact with the duodenal mucosa, to bring about the production of secretin, a hormone which is carried to the liver and pancreas by way of the blood and stimulates the flow of bile and pancreatic juice.

The amount of acid normally present in the stomach of the breast-fed infant is sufficient to activate the pepsin present so that some peptic digestion is possible; the digestion of the proteins of milk, however, is very incomplete in the stomach, even in the case of breast-fed infants. Small amounts of both casein and lactalbumin are converted into albumoses and peptones, but the greater part of the digestion of protein is completed in the small intestine.

Coagulation of casein by rennin is usually complete in the stomach. The casein curd from human milk is very small and fine, whereas the curd from raw or pasteurized cow's milk is very much larger and tougher and may be a single, large, jellylike curd, almost completely filling the stomach. Large curds entangle in their meshes such bacteria as may be present and remove them from the antiseptic action of the gastric juice. These large, tough curds may be somewhat broken up by the gastric contractions, but even then they cannot pass through the pylorus as readily as the finer curds of human milk. Boiling of cow's milk so changes the character of the casein that it is precipitated in smaller curds in the stomach. Heating cow's milk to a still higher temperature or for a longer

period still further alters the character of the casein so that the curds are relatively fine and soft. Thus both evaporated and dried milks tend to produce small curds in the stomach. When cow's milk is appropriately acidified previous to feeding, the casein curds are precipitated in finely divided form and very little if any further change is effected through the action of the rennin of the stomach. Addition of alkalies or of sodium citrate to milk before feeding, or dilution of the milk with water or especially with starch gruels, results in the formation of smaller curds in the stomach. The size of the curds produced in the stomach is an important factor in the digestibility of milk mixtures.

Mention has been made of the fact that the stomach contents of the normal infant receiving human milk are distinctly acid. When equivalent amounts of undiluted sweet cow's milk are fed, the stomach contents do not reach the same degree of acidity, the average concentration of acid at the height of digestion not exceeding a pH of 5.3. This degree of acidity is not sufficient for peptic digestion nor is it sufficient to exert very much inhibitory action on the growth of bacteria. The acidity of the stomach contents passing into the duodenum is also below the optimum for secretin formation. If the feeding of undiluted sweet cow's milk is continued, there occurs a gradual increase in the concentration of acid secreted. Some normal infants secrete such a concentrated gastric juice that even when they are fed undiluted sweet cow's milk the gastric acidity may approximate that of normal breast-fed infants.

The difference in acidity between cow's milk and human milk during gastric digestion is explained by the higher buffer content of cow's milk. Buffers are substances capable of resisting a change in reaction of the medium when either acids or alkalies are added. Cow's milk contains approximately three times as much buffer substance as human milk, so that three times as much acid must be added to a given amount of cow's milk to bring it to the

optimum acidity for gastric digestion. This difference in the two milks is shown in the titration curves. (See Fig. 6.) These curves show the gradual increase in acidity as varying amounts of diluted hydrochloric acid are added. When one part of cow's milk is diluted with two parts of water, the buffer value of the dilute milk is, of course, only one-third that of the concentrated milk, and such dilute milk shows a titration curve approximating that of human milk.

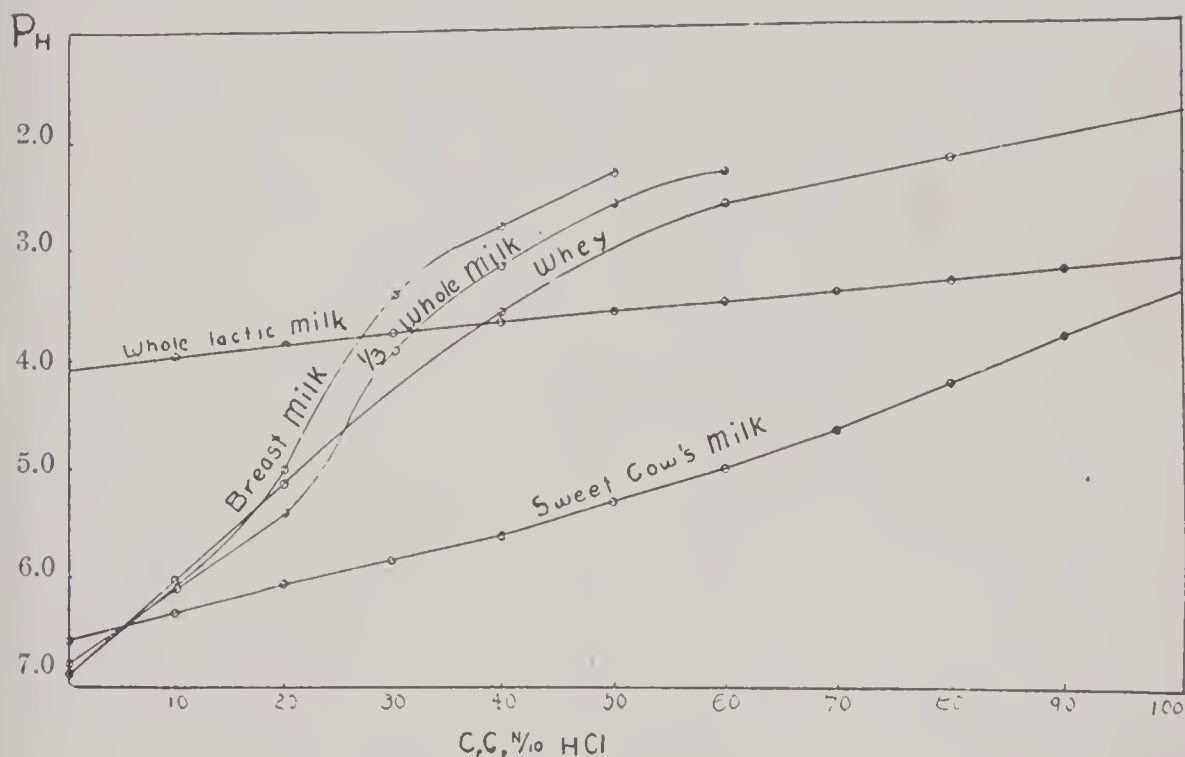


Fig. 6.—Titration curves of human milk, sweet cow's milk, lactic acid milk, cow's milk diluted to one-third, and whey. These curves show the hydrogen-ion concentration resulting when varying amounts of hydrochloric acid are added to milk. The hydrogen-ion concentration is measured in pH units, as shown by the figures at the left. A pH of 7.0 indicates a neutral solution; the *lower* the pH value, the *greater* the degree of acidity. The pH of a solution represents the negative exponent of the hydrogen-ion concentration.

When cow's milk diluted to one-third is fed, the gastric contents at the height of digestion average the same as in the case of human milk. Dilution, therefore, is one of the means of rendering cow's milk more readily digestible. Another method is acidification to neutralize the buffer substance. Acidification is discussed in Chapter XVI.

No appreciable digestion of fat occurs in the stomach, although a small amount of lipase is occasionally present.

Carbohydrates are not acted upon by the gastric juice except for possible slight conversion of disaccharides to monosaccharides at the temperature and acid reaction of the stomach. The ptyalin of swallowed saliva may continue to act in the stomach for a short time on any starch present in the food.

Very little absorption takes place from the stomach and that mostly from the pyloric end. Small amounts of dextrose, amino acids, or salts may be absorbed, but very little if any of other substances. Water is probably not absorbed except in cases of extreme dehydration.

The stomach contents begin to empty through the pylorus during the time that feedings are being taken. The pylorus opens and closes intermittently, allowing but small quantities to pass through at one time. The stomach thus acts as a protective mechanism, tending to prevent the flooding of the intestinal tract with food in excess of the digestive capacity. The length of time required for the complete emptying of the stomach is variable, and dependent on the size of the meal, the character of the feeding and the tonicity of the pyloric and gastric musculature. In general, the stomach empties more quickly in the case of breast-fed infants, being empty usually by the end of two hours. In artificially fed infants, the emptying time may be prolonged to three hours or more.

Dilute milk mixtures are emptied more rapidly than concentrated ones, and small feedings more rapidly than large. Cow's milk which has been treated so that the curds are very small leaves the stomach more quickly than milk which forms large curds. Acidified milk leaves the stomach more rapidly than unacidified. The more liquid portions of the stomach contents are the first to pass through the pylorus. Sugars leave the stomach before proteins and fats. An excess of fat in the food delays the emptying of the stomach. In the presence of fever, gastric motility is sluggish; it is also sluggish in undernourished infants. When

any degree of pylorospasm or pyloric stenosis exists, the emptying time of the stomach is prolonged. If the infant is hungry at the time of the feeding, gastric motility is more active than if the infant is fed at such short intervals that the pangs of hunger are not present. The stomach empties more rapidly if the infant lies on the right side or is held in a semierect position.

Intestinal Digestion

When the chyme passes through the pylorus into the duodenum, it remains for a short time before passing along toward the jejunum. While in the duodenum, the food becomes mixed with pancreatic juice, bile, and some succus entericus.

The pancreatic juice is strongly alkaline in reaction and contains the enzymes trypsin, rennin, amylase (pancreatic diastase) maltase and lipase (steapsin) capable of acting on proteins, carbohydrates, and fats, respectively.

The bile is neutral or slightly alkaline and contains, besides pigments, cholesterol and lecithin, and the salts of the bile acids (glycocholic and taurocholic) which latter are effective in promoting the emulsification of fats. The bile also contains a substance which accelerates the action of pancreatic lipase. Bile is capable of dissolving fatty acids and to a certain extent the soaps of calcium and magnesium which are ordinarily insoluble in water.

The intestinal juice or succus entericus is alkaline in reaction and is secreted by the glands throughout the small intestine. It contains the enzyme enterokinase, capable of increasing the action of trypsin peptidases (erepsin), which complete the hydrolysis of protein fragments and an enzyme or group of enzymes capable of converting the disaccharides lactose, sucrose, and maltose into monosaccharides.

The combined secretions of the duodenum although alkaline in reaction do not normally neutralize completely

the acid chyme coming from the stomach, so that the duodenal contents as well as the contents of the upper part of the jejunum are weakly acid in reaction. When gastric secretion is decreased, or when foods of a high buffer content are fed (such as undiluted sweet milk), the duodenal contents may be alkaline. When the duodenal contents are alkaline, bacteria are likely to be present in large numbers.

Protein digestion, which may occur to a slight extent in the stomach, is continued in the duodenum through the action of the enzyme trypsin of the pancreatic juice, which breaks the protein molecule into smaller remnants, the peptones, polypeptides and amino acids. Protein digestion is further continued by the action of the peptidases, which convert to amino acids those remnants of the protein molecule which have escaped tryptic digestion. It is almost entirely as amino acids that the end products of protein digestion are absorbed. Very small amounts of undigested protein, especially egg protein, may be absorbed as such during the first few months of life. Some globulin from the colostrum is also absorbed during the first week. In the presence of severe diarrhea, the intestinal tract becomes somewhat more permeable to proteins and intermediate products of protein digestion.

Protein digestion and absorption during infancy are remarkably complete, even in the presence of diarrhea and in the case of sick or poorly nourished infants, only an insignificant amount of protein or of protein derivatives appearing in the stools. Such protein as is present in the stools represents chiefly secretion from the intestinal mucosa together with bacterial proteins.

The simple carbohydrates, dextrose, levulose, and galactose, require no digestion and are rapidly absorbed by the small intestine. The disaccharides, maltose, sucrose, and lactose, are converted into the monosaccharides through the action of enzymes secreted by the small intestine and are absorbed in the form of monosaccharides. When a

large amount of carbohydrate is fed, or when absorption is impaired, some sugar may reach the large intestine and here be attacked by the bacteria present. Sugar itself rarely appears in the stools; it decomposes to form acids and gases.

Starch digestion is effected by the enzyme amylase of the pancreatic juice. Amylase has the same action as the ptyalin of the saliva or the diastase of germinated grain. It converts starch first to dextrin and then to maltose. Amylase is present in the pancreatic juice of the young infant in only small quantities. When starch is fed, however, the secretion of amylase appears to be stimulated so that even a young infant accustomed to the feeding of starch may digest a considerable amount. Relatively little carbohydrate is normally destroyed through bacterial action in the intestinal tract. If, however, the digestive and absorptive capacity of the gastrointestinal tract is impaired, bacterial destruction of a large portion of the carbohydrate may occur. The products of bacterial action on carbohydrate are chiefly organic acids, some of which have a distinctly irritant effect on the intestinal mucosa. Excessive fermentation of carbohydrate results in impaired absorption not only of carbohydrates but also of other food components.

The digestion of fats begins in the duodenum, the first step being emulsification or the breaking up into very fine globules. This process is aided by the presence of bile salts and by the churning action in the duodenum. Milk fat is already in a fine emulsion, but other fats require emulsification before much digestion is possible. The next step in the digestion of fats is saponification, which is accomplished by the lipases of the pancreatic and intestinal juices. The activity of these enzymes is enhanced by the presence of bile. Through the action of the lipases, the emulsified fat is converted into fatty acids and glycerol and absorbed as such. When the duodenal contents are alkaline or when

the partially digested fat reaches the lower portions of the small intestine where the reaction of the contents is alkaline, soaps are formed by the combination of fatty acids with the bases present. Some calcium soaps also are formed, especially when the food contains a good deal of casein (calcium caseinate). These calcium soaps are partially dissolved by the bile and absorbed, the remainder pass through the intestinal tract and appear in the stools as curds or as solid, puttylike masses.

The fat of the food, whether from human or cow's milk, is almost completely saponified under normal conditions. Absorption, however, is more nearly complete in the case of human milk fat. In the presence of diarrhea, the intestinal contents may be hurried through so rapidly that some fats escape saponification, appearing in the stools as neutral fat. When the flow of bile into the intestinal tract is obstructed, saponification and absorption of fats are not complete and large amounts of soaps and some neutral fat pass through the intestinal tract unutilized. In the condition of celiac disease (see Chapter XXII) saponification of fats is usually complete, but absorption is extremely poor, and a large portion of the ingested fat may appear in the stools in the form of soaps.

In general, infants digest and absorb fats far less efficiently than they utilize either proteins or carbohydrates. Even a slight digestive disturbance is likely to result in diminished absorption of fat. Unabsorbed fatty acids, particularly the lower members of the series, are capable of causing gastrointestinal irritation.

The large intestine secretes an alkaline juice, but apparently no digestive enzymes. Any digestion that occurs in the large intestine is merely a continuation of digestive processes in such mixtures of food residues and enzymes as have previously escaped absorption.

In the large intestine bacterial activity is usually vigorous and results in the decomposition of certain food rem-

nants, especially carbohydrates. Under normal conditions more absorption of water probably occurs from the large than from the small intestine. The contents of the small intestine are normally liquid; those of the lower portion of the large intestine, semisolid. Very little absorption of food materials occurs from the large bowel, though the colon is capable of absorbing amino acids, monosaccharides, salts, and water.

Bacteriology of the Gastrointestinal Tract

At birth the gastrointestinal tract is sterile, but it does not remain so for long. The bacterial flora differs greatly in the different portions of the intestinal tract, and the number and character of the organisms present are influenced profoundly by the character of the diet, by the nature and amount of the gastrointestinal secretions, and by the occurrence of fever. Almost any organism present in the air, in water, or in milk may at times be found in the gastrointestinal tract. The most important and most frequently found organisms are *Lactobacillus bifidus*, *Aerobacter aerogenes*, *Lactobacillus acidophilus*, *Escherichia coli*, and *Streptococcus faecalis*. Occasional inhabitants of the intestinal tract are *Clostridium perfringens* (the gas bacillus), staphylococci, and streptococci. Certain other organisms, such as the members of the dysentery group, typhoid, paratyphoid, tubercle bacillus and *Bacillus lactis* (bacillus of Flügge), are occasional invaders of the intestinal tract and give rise to disease.

Under normal conditions, considerable inhibition of bacterial growth is brought about through the acidity of the gastric juice, so that the food entering the small intestine is relatively free from bacteria. The gastric acidity is sufficient to inhibit the growth of *Escherichia coli*, the hemolytic streptococci, enterococci and many other organisms. Most spore bearers and anaerobic organisms are little affected by the gastric juice. The upper portion of the small intestine usually contains relatively few organ-

isms, those present being chiefly cocci and a few members of the *Aerobacter aerogenes* group. When the gastric secretion is decreased, however, as the result of fever or high external temperatures, or is neutralized by the giving of an excess of alkali or of large amounts of sweet cow's milk with its high buffer value, the upper portion of the intestinal tract may contain large numbers of bacteria. Colon bacilli are often found in the stomach and duodenum in the presence of severe gastrointestinal disturbances with diarrhea. Whether these organisms have ascended from the colon as the result of gastrointestinal disturbance, or whether they have been introduced in the food and having found suitable conditions for growth have, by their activity, brought on the diarrhea, is an unsettled question. It is our own belief that this invasion of the upper intestinal tract by colon bacilli and related organisms is an important factor in the causation of diarrhea.

Growth of microorganisms in the upper intestinal tract is favored by an excess of food which furnishes a suitable culture medium. It is also favored by damage to the intestinal mucosa or by decrease in the normal digestive secretions. The type of organisms which predominate is determined, to a large extent, by the character of the food. Some bacteria thrive in a medium rich in carbohydrate; others grow better in the presence of protein.

In the lower portion of the small intestine bacteria are more numerous, *Lactobacillus bifidus*, *Streptococcus faecalis*, *Escherichia coli*, *Lactobacillus acidophilus*, and *Aerobacter aerogenes* predominating.

In the large intestine enormous numbers of bacteria are normally present. In the case of breast-fed infants, the predominating organism present in the lower bowel is the *Lactobacillus bifidus*, a gram-positive acid-producing organism. *Escherichia coli* and *Lactobacillus acidophilus* are found in smaller numbers, and occasionally *Clostridium perfringens* is present. *Streptococcus faecalis* may be pres-

ent in large numbers. In the colon of the artificially fed infant the flora is more complex, gram-negative bacilli of the *Escherichia coli* and *Aerobacter aerogenes* groups predominating. The feeding of considerable amounts of lactose together with relatively small amounts of protein tends to cause an increase in the number of *Lactobacillus bifidus* present. In the large intestine such carbohydrates as have escaped digestion are fermented and converted into gases and organic acids. Protein remnants are also broken down by bacterial action to form ammonia, amines, indol, skatol and a variety of other products.

The growth of bacteria in the intestinal tract may either aid or interfere with the processes of digestion. The growth of such organisms as the *Lactobacillus acidophilus* and other harmless lactic-acid producers in the upper portion of the intestinal tract brings about a condition of acidity which inhibits the growth of certain potentially harmful organisms. A moderate degree of acidity also promotes the absorption of calcium salts. Other organisms which ferment carbohydrates or decompose fats with the production of large amounts of acetic, formic and other organic acids exert a harmful influence, as certain of these acids are irritating to the intestinal tract.

Some strains of *Escherichia coli*, *Clostridium perfringens*, and certain streptococci growing under favorable conditions produce large amounts of amines, such as histamine and tyramine. Under normal conditions, histamine is detoxified on passage through the intestinal mucosa and the liver, so that it does not exert a harmful effect. Apparently considerable amounts of histamine can be produced in the colon without causing damage to the body as a whole. When, however, histamine is injected subcutaneously, or is absorbed from the mucosa of the mouth, very toxic symptoms are produced which are of a shocklike nature, often accompanied by vomiting, diarrhea, and anhydremia. There is evidence that histamine and similar

substances may be absorbed through an injured intestinal mucosa and not be completely detoxified. It appears likely that the growth of histamine-producing organisms in the upper portion of the small intestine is capable of producing severe gastrointestinal disturbances. It has been shown, furthermore, that when the contents of the upper portion of the small intestine are strongly alkaline, colon bacilli may pass through the mucosa and enter the blood stream.

The *Clostridium perfringens* (the gas bacillus) decomposes sugar with the formation of hydrogen and butyric acid. This latter is irritating and may, when present in large amounts, lead to diarrhea.

CHAPTER X

THE STOOLS IN INFANCY

Meconium

The first stools of the infant consist of dark, brownish green, semisolid meconium. The first passage is likely to occur during or very shortly after birth. The meconium consists of partially dried intestinal secretions which have accumulated in the large intestine from the fourth fetal month on. The amount of meconium thus accumulated is usually sufficient to fill the distended sigmoid and descending colon and amounts to from 5 to 7 ounces (150 to 200 grams). The gradual passage of meconium during the first three or four days of life accounts for much of the initial weight loss of the newly born infant. The meconium is usually passed from three to five times daily. By the third or fourth day, with the ingestion of milk, there occurs a gradual transition to the usual type of infant stool. In cases in which the food intake is very small, the meconium-like character of the stools persists for a longer period.

Stools of the Breast-Fed Infant

The normal breast-fed infant usually passes from two to four stools a day. These stools are soft and golden yellow or greenish-yellow in color, acid in reaction, and have a slightly sour, but not unpleasant odor. The number of stools is subject to considerable variation. Some perfectly normal infants who are thriving may have only one stool a day, and others as many as seven or eight. The consistency also may vary from semiliquid to salvelike. The color may vary from yellow to definite green. Any sudden change in the character or number of the stools is of significance.

Stools of the Artificially Fed Infant

Infants fed cow's milk usually pass a smaller number of stools than those who are breast fed. The stools are, however, more bulky and contain more solid material. The color differs from that of the human-milk stool, being lighter yellow or grayish brown. The reaction is usually either neutral or alkaline, as compared with the acid reaction of the human-milk stool. The odor is more unpleasant, especially when relatively large amounts of milk and small amounts of sugar are fed. When foods other than milk and sugar are added to the diet, the character of the stools changes until, with a general mixed diet, the stools are of the same character as those of the adult.

The Number of Stools

The number of stools depends on the total amount and character of the food, on the presence or absence of infection in the intestinal tract or elsewhere, and on the individual tonicity of the gastrointestinal musculature. A large proportion of fermentable carbohydrate in the food, especially lactose, leads to an increase in the number of stools. An excess of protein, especially casein, leads to a condition of alkalinity in the intestinal tract and decreased peristalsis, so that the stools are passed less frequently. An excess of fat, together with an excess of carbohydrate, leads to frequent movements, but a relatively large amount of cow's milk fat, together with considerable protein and minimal amounts of fermentable carbohydrate, sometimes leads to constipation with the passage of large, firm, soap stools. An insufficient intake of food may result in constipation but is as likely to lead to the passage of numerous small, dark green stools—the so-called starvation diarrhea.

Infestation of the intestinal tract, especially the upper intestine, with bacteria capable of decomposing food with the production of irritating products leads to the passage of numerous liquid stools. Dysentery infections causing

ulceration in the lower intestinal tract result in the passage of very numerous stools containing mucus, pus, and often blood. Infections outside the intestinal tract also may lead to diarrhea. The influence of such infections is further discussed in Chapters XX and XXVIII.

Some infants have constitutionally atonic intestinal musculatures, and in consequence sluggish peristalsis, and suffer from constipation. Other infants are constitutionally of the hypertonic type, have active peristalsis, and suffer from a tendency to diarrhea.

The Color of the Stools

The color of the stools depends chiefly upon the character of the diet and the rate of peristalsis. Bile pigment gives the predominating color to most stools. The bile as secreted in the upper intestine is green, due chiefly to biliverdin. When peristalsis is active, the bile, mixed with the food, passes through the intestinal tract largely unaltered, and the stools are consequently green in color. When food remains in the intestinal tract for a longer period of time, reduction of the green biliverdin to yellow or brown bilirubin occurs. When the contents remain in the intestine for a still longer time, bilirubin may be reduced in part to colorless hydrobilirubin. The stools are then very light colored. The presence of oxidizing bacteria in the intestinal tract favors oxidation of yellow bilirubin to green biliverdin, especially in the presence of acid. The stools, under these conditions, are green. These facts explain the green color of certain diarrheal stools, the yellow color of the stools of the normal breast-fed infant when moderate peristalsis is present, and the light color of the constipated stools of the artificially fed infant. A stool which is yellow when passed may change to green on exposure to the air due to oxidation of bilirubin. Diminished or absent secretion of bile results in lack of color of the stools. A large proportion of calcium soaps in the stools also causes them to be light in color. When cereals and certain malt prepara-

tions are fed, the stools assume a brownish shade. Occasionally a pinkish ring is seen at the edge of stools which have remained for some time on the diaper; this color is usually due to altered bile pigment, or occasionally to urates; it is without significance, but may be mistaken for blood.

When spinach is fed, the stools may be green, and the feeding of tomatoes or beets may cause occasional red specks in the stool. Bismuth, iron, or argyrol may color the stools dark brown or black. Blood which has been swallowed or passed high in the intestinal tract also will color the stools dark reddish brown or black. Chemical tests may be necessary to differentiate blood coloration from that caused by drugs. Small streaks of blood on the outside of a constipated stool are generally indicative of anal fissures. Bright blood mixed with the stool is usually indicative of ulceration in the colon or lower ileum and is seen characteristically in the presence of bacillary dysentery. Large amounts of bright blood appear soon after intussusception.

Curds

The solid material of the stool consists largely of calcium soaps, calcium phosphate, mucus, and dead bodies of bacteria. When raw milk is fed, casein curds also may be present. When peristalsis is sluggish, a good deal of the water from the intestinal contents is absorbed, and the solid material of the stool is compressed into firm masses consisting largely of calcium soaps. When peristalsis is active, less water is absorbed, and the churned-up calcium soaps appear in the stools as soft, white, or bile-stained curds. The presence of such curds indicates merely that fat has been present in the diet and that peristalsis has been active. Soap curds are often found in the stools of normal breast-fed infants and are of little significance.

In the stools of infants fed raw or pasteurized cow's milk, hard, yellow, beanlike masses of undigested casein may occur. These casein curds are not seen in the case of

infants fed human milk or boiled, evaporated, dried, or acidified cow's milk.

Small stringy curds or partially dried balls of mucus are occasionally seen, especially in the stools of breast-fed infants.

Mucus

A small amount of mucus is present normally in the stools. The amount is increased whenever there is intestinal irritation. Especially large amounts of mucus occur in the presence of bacillary dysentery. The mucus is secreted chiefly by the mucosa of the lower bowel.

Starch

When young infants are fed large amounts of starches or cereals, a portion of the starch escapes digestion and appears in the stools, giving them a slimy consistency. When such stools are brought into contact with iodine solutions, the starch particles are stained a deep blue. The passage of undigested starch through the intestinal tract rarely causes harm, but occasionally excessive fermentation of starch occurs in the intestine, and it then becomes necessary to reduce this constituent of the diet.

Cellular Elements

A few leucocytes and epithelial cells may be found microscopically in most stools. Any great excess of pus cells sufficient to cause the appearance of macroscopic pus is abnormal and is indicative of intestinal lesions. The appearance of pus in the stools is, in most cases, indicative of intestinal ulceration or dysentery. Red blood cells do not appear normally in the stools.

Small pieces of membrane or large numbers of desquamated epithelial cells are most frequently seen in the earlier stages of bacillary dysentery. Membrane-like casts of the bowel, several inches long, may be seen in cases of membranous colitis, a rather uncommon hereditary disturbance of the intestinal glands.

Bacteriology of the Stools

Bacteriological examination of the stools gives definite information concerning the bacteriology of the lower bowel only and does not necessarily serve as an indication of conditions in the upper intestine. Bacteriological examination of the stools is of especial value in arriving at a diagnosis of bacillary dysentery. For technique, see Chapter XXXII. Some pediatricists consider the finding of the gas bacillus (*Clostridium perfringens*), in the stools to be of pathological significance. This organism is frequently found, even in normal stools, but when very large numbers are present, the products of fermentation, especially butyric acid, may possibly cause irritation of the mucosa and consequently diarrhea.

In the alkaline soap stools of artificially fed infants, the *Alcaligenes ammoniagenes* (*B. ammoniagenes*—Cooke) is not infrequently present. This organism is capable of decomposing urea with the formation of ammonia. The presence of this organism is the cause of the “ammoniacal diaper” and resultant dermatitis so often observed in the case of artificially fed infants. It has been shown by Cooke that rinsing of the diapers in 1:5,000 bichloride of mercury or in saturated boric acid solution before drying results in the inhibition of the growth of this organism in the urine of the wet diaper. If boric acid is used, it is desirable to sprinkle additional boric acid powder in the diaper when it is applied. Changing the character of the diet by reducing protein and fat and increasing carbohydrate so as to cause an acid condition of the contents of the lower bowel also results in inhibition of the growth of *Alcaligenes ammoniagenes* as it thrives only in an alkaline medium. Changes in the diet, however, are not necessary in the treatment of the condition and usually are contraindicated. “Ammoniacal diaper” is the only common cause of meatal ulcer in circumcised male babies.

Significance of Stool Examination

From what has been said, it is evident that the stools may vary greatly even under normal conditions. If an infant is gaining in weight, has no fever, and is healthy in appearance, it makes very little difference whether the stools number one or seven a day, whether they are firm or semisolid, whether they are light yellow, orange, or green, and whether or not they contain fat or protein curds. The general health of the infant is of far more significance than the character of the stools. If, however, an infant who has normally been having one or two stools a day begins to have six or seven with an increased amount of mucus, and if the stools become watery, the probability is that some infection either within or without the gastrointestinal tract has occurred or that some unsuitable article of diet has been taken. Very large, light-colored, foul stools containing much soap and some neutral fat are seen in cases of chronic intestinal indigestion or celiac disease. The presence of macroscopic pus and blood in the stools is indicative of definite abnormal conditions. Microscopic examination of the stools of infants usually gives little information that cannot be obtained by simple inspection.

CHAPTER XI

BREAST FEEDING OF THE NORMAL INFANT

General Considerations

The milk of a healthy mother who is receiving a diet nutritionally adequate in all respects may be expected to contain all the nutritional factors necessary for the infant, and in sufficient amount, with the exception of iron, vitamin D, and possibly thiamine. Presumably Nature intended that vitamin D be obtained from radiant energy. The iron stored in the body at and after birth may be expected to compensate, during the first six months of life, for the low iron content of milk, if the baby remains in good health. The caloric value of human milk is such that when the infant is fed at reasonable intervals and takes as much as he desires, the total energy requirements are met. Little danger of over- or underfeeding exists provided nursings are at proper intervals, and the mother's supply of milk is adequate. The milk in the breasts is normally free from harmful bacteria, and if reasonable cleanliness is observed and the nipples do not become infected, the milk as received by the infant will also be free from harmful bacteria. Volume for volume, human milk is more readily digested by the human infant than is undiluted cow's milk. It has been assumed that human milk contains certain immune bodies which are of value in rendering the infant resistant to infection, but convincing proof of this fact is lacking and furthermore most immune bodies are destroyed during the processes of digestion. It has been shown in the case of animals that absorption of globulins from the colostrum during the first few days of life is a factor in increasing immunity; it is not certain whether or not this is the case in human beings.

Infants who are breast fed by healthy well-nourished mothers with abundant milk secretion are usually healthy babies and have fewer infections than artificially fed infants. The mortality during the first few months, according to most statistical studies, is lower among infants who are entirely breast fed.

One of the important factors in favor of breast feeding is the emotional satisfaction which both the mother and the baby derive from the process. The baby gets a sense of security and satisfies his hunger without the emotional disturbance of the mother that often occurs if the baby refuses part of his bottle feeding, or demands more than his rationed portion. The mother sits in a comfortable chair holding her infant while he feeds. She gets rested and the immeasurable satisfaction that goes far in establishing good parent-child relationship. With well-chosen supplementary foods the breast-fed baby thrives well until he is old enough to eat his food at meals.

Because a baby is breast fed, however, does not insure that he is always well fed. The mother's milk may be deficient in quantity, in quality or in both. The amount and quality of human milk depend in large measure on the mother's diet and health. The vitamin content of the milk in particular is directly dependent on the intake of the mother. Vitamin deficiencies have been observed in breast-fed infants. Also some mothers secrete dilute milk, so low in protein, calcium, and phosphorus that the baby cannot get enough of these food factors in the volume of milk he can ingest. A baby getting too little milk may not complain. Unless the baby's weight is measured regularly, the mother may be unaware that her infant receives insufficient food. Notwithstanding all these facts, it is still advisable to have a baby feed at the breast whenever possible, and it is possible at least for the first few months in over 85 per cent of cases. Breast feeding should be encouraged especially among the poor, the ignorant, and the careless, for it is difficult for these people to carry out

satisfactory artificial feeding. The total amount of food given may not be sufficient, certain essentials may be omitted, and bacterial contamination occurs frequently if artificial feeding is attempted. The deficiencies of artificial feeding as practiced by these mothers far outweigh those of breast feeding.

Many babies are weaned unnecessarily because of minor complaints. On the other hand, it is not advisable to allow a baby's nutrition to suffer because the mother has an insufficient supply of milk, or to urge a delicate mother, who has insufficient milk, to nurse an infant who is in good health and capable of thriving with an artificial formula.

Contraindications to Breast Feeding

Certain definite contraindications to maternal nursing exist. A mother with active tuberculosis should not nurse her baby, nor should she come in contact with it in any way. Tuberculosis is probably not transmitted directly through human milk, but the close contact with the mother necessitated by nursing exposes the baby to infection, and young infants are extremely susceptible to tuberculous infection. We have seen a young infant succumb to tuberculosis who had been handled only twice by a mother in the active stage of the disease.

A mother who is herself in poor general health and suffering from severe chronic illness such as advanced nephritis, cardiac disease, or cancer should not nurse her infant. It has been demonstrated in cancer-inheriting strains of animals that an agent which may produce malignant growths is transmitted through the milk during the pre-cancerous stage of the mother. In the present stage of our knowledge of cancer production, it is appropriate that mothers with family history of cancer refrain from breast feeding their infants. A mild degree of nephritis without uremia or nonprotein nitrogen retention is not a contraindication to nursing. Diabetes is usually not a contraindi-

eation; in fact, the mother's carbohydrate tolerance is higher during the nursing period because of the secretion of carbohydrate in the milk.

In the presence of mastitis, use of the affected breast should be discontinued. With double mastitis, complete weaning is necessary. Acute infections on the part of the mother necessitate *temporary* weaning, but the breasts should be emptied by manual expression (for technique see p. 180) and the infant should be put to the breast again as soon as the mother's temperature has returned to normal. More prolonged infections, such as typhoid, necessitate permanent weaning. If the mother has to undergo a surgical operation, the infant should be taken from the breast temporarily and the breasts pumped until after the mother has recovered from the effects of the anesthetic.

Syphilis in either the mother or the baby is no contraindication to nursing. The nutrition of the syphilitic infant is likely to be poor, even under the best circumstances, and it is these infants who especially require mother's milk. Certain infants, especially those born prematurely, are too weak to nurse satisfactorily; others are unable to nurse because of such deformities as cleft palate and hare-lip. Under such circumstances, the breasts should be emptied mechanically and the milk fed to the infant from a bottle or medicine dropper.

The occurrence of menstruation during the period of lactation is not an indication for weaning. When pregnancy occurs, it is advisable to wean the infant gradually, but many mothers are able to nurse their babies satisfactorily during the first few months of pregnancy, provided they are themselves in good health. It has been claimed that continuation of nursing during the latter half of pregnancy tends to cause miscarriage.

Vomiting, colic, diarrhea, or failure to gain in weight is not necessarily indication for weaning. Such symptoms may call for more frequent or less frequent feeding, for

attention to the diet and hygiene of the mother, or, occasionally, for complementary or supplementary feedings from the bottle.

CHARACTERISTICS OF HUMAN MILK

Colostrum

During the first few days post partum the milk secreted differs considerably in character from that secreted later. The first milk, known as colostrum, is a thin, yellowish fluid containing more protein and salts and less fat and sugar than the later milk. It contains numerous large lymphocytic cells—the “colostrum corpuscles.” Colostrum has also a much higher content of some vitamins than does milk secreted later. Unlike milk, colostrum is readily coagulated by heating. It appears to have a certain laxative effect and may aid in bringing about evacuation of the meconium. Experimental work on animals indicates that globulin is present in colostrum which may be absorbed unchanged into the blood and facilitate the development of immunity. It has not been shown that such transfer occurs in the case of the human infant.

The amount of colostrum secreted is relatively small, usually under 100 ml. in the course of a day. About the third or fourth day the milk “comes in.” The breasts become distended and tender. Secretion increases sharply in amount and the milk gradually assumes its mature character. For at least one or two weeks, however, the milk still retains some of the characteristics of colostrum. By the end of the first month, the character of the milk is essentially the same as that which is secreted during the remainder of the nursing period.

Composition of Human Milk

The chief constituents of human milk are fat, sugar, protein, mineral salts, water, and vitamins. Other substances of uncertain composition also are present, the functions of which are not fully known. The average composition of

mature human and cow's milk is shown in Table IV. Human milk is much more variable in composition than is generally realized. Samples of milk from different women may show a range of more than 100 per cent in the amounts of each constituent. For milk of good quality, ingestion of a diet adequate in all respects is essential. The following discussion concerns "average" human milk.

The caloric value of human milk is approximately 650 calories to the liter, or 20 calories to the ounce.

The amount of fat in human milk is more variable than that of either protein or carbohydrate. Although the usual variation is from 2.5 to 5.0 per cent, values as low as 1.0 per cent and as high as 10 per cent have been observed. The first portions of milk removed from the breast are low in fat; the last portions, or strippings, considerably higher. No conclusions as to the fat content of the milk should be drawn unless the breast is entirely emptied and the fore and last portions mixed.

The fat of human milk is present as minute globules held in a state of emulsion. The chief fats are tripalmitin, tristearin and triolein, of which triolein comprises about 35 per cent. About one-half the fats are unsaturated. Human milk fat contains about twice as much of the fatty acids of the so-called essential group and much less volatile fatty acids than cow's milk. This latter fact is of some importance in infant feeding, as the volatile fatty acids are somewhat irritating to the gastrointestinal tract.

The mean value for protein content of human milk, as listed by various investigators, varies from 0.78 to 2.1 per cent. The variation in individual milks must be much greater. The protein content of colostrum is usually much greater than that of milk secreted later. Values up to 5 per cent are reported for colostrum. The proteins of human milk are casein, lactalbumin, and lactoglobulin, 60 per cent (nearly two-thirds) of the total protein present being lactalbumin. The characteristics of these proteins are discussed elsewhere (see Chapters III and IX).

TABLE IV
COMPARATIVE ANALYSES OF HUMAN AND COW'S MILK
PROXIMATE ANALYSIS

A. Values in Gm. per 100 Gm. or ml.										
	CALORIES	FAT	LACTOSE	TOTAL PROTEIN	LACT-ALBUMIN-	CASEIN	TOTAL ASH	WATER		
Human	65-70	2.8-4.1	6.8-7.8	0.8-2.1	0.5-1.0	0.3-0.8	.21-.25	87-90		
Cow's	65-70	3.4-5.0	4.5-5.2	3.1-3.8	0.5	3.0	0.6-0.8	85-88		
B. Minerals, Values in Gm. per 100 Gm. or ml.										
	Ca	Mg	K	Na	P	S	Cl	Fe	Cu	F
Human	.032	.004	.041	.011	.013	.014	.032	.1	.03	
Cow's	.118	.012	.155	.050	.093	.033	.105	.5	.02	.012
C. Vitamins, Values per 100 Gm. or ml.*										
	(TOTAL)		PANTOTHENIC		CHOLINE		BIOTIN		PYRIDOXINE	
	I.U.	MG.	MG.	MG.	MG.	MG.	MG.	MG.	MG.	I.U.
Human	290	.014	.037	.183	.246	.0008	.0008	.004	5.0	5
Cow's	180	.038	.200	.085	.350	14.9	.003	.067	1.0	2
D. Some Amino Acids, Values in mg. per 100 ml.†										
	ARGI-NINE	PHENYLALANINE	LEUCINE	ISO-LEUCINE	HISTIDINE	LYSINE	THREONINE	METHIONINE	CYSTINE	TRYPTOPHANE
Human	57	77	228	75	20	75	63	25	30	25
Cow's	127	177	490	167	61	220	151	100	24	45
E. Molar Percentage Distribution of Total Fatty Acids‡										
	PER CENT SAT'D		PER CENT UNSAT'D		SAT'D		PALMITIC		STEARIC	
					C ₁₄	OR LESS			OLEIC	C ₁₈₋₂₂ ACIDS, UNSAT'D
Human	48.8	51.2	51.2	40.9	18.8		24	8.5	33.2	10.5
Cow's	59.1	40.9	40.9		27.4		21.1	10.6	31.4	5.4

*Values for human milk from Macy and associates: Am. J. Dis. Child. 70: 135-192, 1945.

Values for cow milk from Lawrence, Herrington, and Maynard: Am. J. Dis. Child. 70: 193, 1945.

Additional values for human and cow milk, respectively; inositol .033 and .018 mg., folic acid .045 and .005 mg., vitamin K, 2 and 0.5 Dam-Glavind units. Vitamin A (preformed) .065 and .033 mg., carotene .025 and .030 mg.

†From Beach and associates: J. Biol. Chem. 139: 57, 1941, and Williamson: J. Biol. Chem. 156: 47, 1944. Cystine S and methionine S, 9.1 and 28.3 mg. per 100 ml. for human and cow's milk, respectively.

‡From Hilditch and associates: Biochem. J. 38: 29, 1944, and Longenecker and associates: J. Biol. Chem. 154: 255, 1944.

The amount of protein in human milk is much less than in cow's milk, but because of the higher proportion of lactalbumin, it has a good nutritional value. Lactalbumin is a complete protein and has been shown superior to casein in promoting growth and in correcting hypoproteinemia of animals; some evidence indicates that it may not be superior for the human. The amino acid content of human milk may be compared with that of cow's milk diluted one-half, so as to approximate the same total protein content. Such a comparison is valid, as cow's milk is often fed in this dilution, especially to very young infants. The essential amino acids with the exception of tryptophane, methionine, and cystine are found in approximately equal amounts in human and the diluted cow's milk. The diluted cow's milk contains only one-third as much cystine as human milk but 1.5 times as much methionine. Cystine, while not truly an essential amino acid, has a sparing effect on methionine need. According to Williamson, the total sulfur-containing acids are approximately equal in the two milks (0.36 and 0.38 millimols for human and diluted cow's milk, respectively). Human milk has an ample amount of tryptophane, whereas the diluted cow's milk is definitely inferior in its content of this amino acid. However, undiluted cow's milk contains ample amounts of all necessary amino acids. The amount of protein in human milk usually is sufficient for the needs of the average normal full-term infant. It is not enough for the needs of the prematurely born or greatly undernourished infant, and supplementary protein must be given.

The sugar of human milk is exclusively lactose. The amounts present are remarkably constant, varying less than those of any other constituent. Such variations as are reported in the literature are to be explained largely on the basis of the different analytical methods used.

Human milk of good quality contains all the mineral salts necessary for the nutrition of the normal infant dur-

ing the first few months. However, it does not supply enough of all the minerals to meet the needs of the infant after the fifth or sixth month, so that mineral-containing supplements should be given. The mineral content is much lower than that of cow's milk, but the utilization of all minerals is excellent. Although human milk contains somewhat more iron than cow's milk, the amount is insufficient for continued hemoglobin formation after the initial iron stores of the body are exhausted. The high content of human milk in folic acid may be a factor in the lesser incidence of anemia in breast-fed babies. The iron, calcium, and phosphate, as well as the protein, provided by human milk are insufficient to meet the needs of the rapidly growing prematurely born infant. For these babies, dried skimmed cow's milk and iron should be added to the human milk. The buffer value of human milk, or the capacity for neutralizing gastric acid, is very low as compared with that of cow's milk.

The content of human milk in the known vitamins is shown in Table IV, *C*. The actual values found in any sample of milk vary with the diet of the mother. Vitamins A, K, C, niacin, folic acid, and inositol are usually found in greater concentration in human than in cow's milk. However, vitamin deficiency can and does occur. Scurvy is uncommon in a breast-fed baby even though no vitamin supplement is given. Rickets is less common than in artificially fed babies, but occurs sufficiently often that an addition of vitamin D to the diet of the breast-fed baby is advisable. Vitamin A deficiency of breast-fed babies has been reported in the Far East, thiamine deficiency in Australia, and niacin deficiency in this country.

The bacteria usually found in human milk are not pathogenic for the healthy infant. Such bacteria as are found are chiefly nonpathogenic cocci derived from the external milk ducts. In the presence of suppurative mastitis the causative organism may be present in the milk in large numbers. Any organism present in the mother as a cause

of septicemia may pass into the milk. Tubercle bacilli are not found in human milk unless the breast is the site of the disease.

The Hygiene of the Nursing Mother

In order that there may be an adequate secretion of milk of good quality, it is essential that the mother herself be in good physical condition and that she receive an adequate diet. The mother should have plenty of sleep and exercise in the open air. If the feeding intervals are not too short, the mother can have more time free for rest and recreation. It is especially desirable that she should have, if possible, unbroken sleep during the night. After the infant is a few weeks old, it is often possible to omit the night feedings. The mother should rest for at least an hour each afternoon. She should relax completely and rest for a while after each nursing.

Any form of outdoor exercise which the mother enjoys is good for her, provided it is not carried to the point of fatigue. A mother who is overworking around the house and is tired out is not likely to be benefited if forced to take additional outdoor exercise. Sitting outdoors in a comfortable chair, or motoring, will be of more benefit in such a case. The mother should, as far as possible, be relieved of worry and mental strain. If her supply of milk is insufficient in quantity and the baby is consequently always hungry and fretful, the mother will get little rest and her milk supply will not increase. Under such circumstances, it is better to give the baby additional food in the form of a suitable artificial feeding.

Amounts of Milk Secreted

The total amount of milk secreted daily depends on the demands of the infant and the capacity of the breasts to secrete. The chief stimulus to milk secretion is complete emptying of the breasts. A healthy, vigorous infant will be likely to empty the breast rather completely, with re-

sultant stimulation of milk secretion. The amount of milk secreted by a healthy mother usually increases in proportion to the infant's demands. Some mothers are able to nurse more than one infant. Regular and complete emptying of the breasts either by the infant or by manual expression may increase the amount of milk enormously. Wet nurses, especially Negroes, have been known to produce more than a gallon of milk a day. It is impossible to state beforehand whether or not a mother will be able to produce enough milk to meet entirely her infant's needs. Some thin, but healthy, mothers with small breasts, produce much larger quantities of milk than large, well-developed fat mothers. A mother who has been unsuccessful in nursing previous infants is not likely to be able to secrete enough milk.

Diet of the Nursing Mother

If the mother's diet is already adequate in every particular, forced overfeeding will not increase the secretion of milk, but the milk secretion of the underfed mother may be increased by the giving of food sufficient in quality and quantity. The amount of human milk secreted in a day to meet the needs of the nursing infant will have a food value of from 500 to 1,000 calories. This extra food must come from the mother's body or be supplied in her diet. The nursing mother consequently should take more food and more fluids than under ordinary conditions. Milk may be secreted, however, even at the expense of the mother's own tissues.

The nursing mother does not require a very special form of diet. A diet that is suitable for her under normal conditions will be suitable during the period of lactation except that she will need to take a somewhat larger amount. There seems to be no good basis for the idea that the nursing mother cannot take acid fruits, vegetables or salads. These are all good for her and will not upset her digestion. Certain foods such as onions and garlic give a

taste to the milk to which older infants may object. The daily diet should contain meat, milk, butter, eggs, two green vegetables, one of which should be raw, and fresh fruit.

Milk is especially valuable as it supplies the materials from which the mother may produce milk. The nursing mother should take from 3 to 4 pints (1500 to 2000 ml.) of milk a day. It is not necessary that she take this in the form of liquid milk. To some adults, milk is distasteful; in such cases the milk may be cooked in the food or served in the form of cocoa, or as ice cream or custards. Evaporated milk lends itself well to the preparation of various dishes and has essentially the same value in nutrition as ordinary bottled milk. One pint of evaporated milk is approximately equivalent to a quart of bottled milk, and this amount can easily be incorporated in the daily diet.

Butter contains the vitamin A of the milk and whatever vitamin D may have been present. Not less than 30 grams (one ounce) should be taken daily. Oleomargarine fortified with vitamin A may be substituted for butter. Full cream cheese is almost as valuable as the milk from which it is made, containing most of the fat, protein, and fat-soluble vitamins, and considerable calcium and phosphorus.

Eggs are valuable because of their content of many of the nutritional essentials. One egg, or preferably two, should be taken daily. These, like milk, may be incorporated in other foods.

Green vegetables should have an especially important place in the diet because of their content of the B group of vitamins, vitamin C, and mineral salts, as well as for their laxative effect. The vitamin B₁ requirements for lactation are high. A leafy vegetable should be served daily as salad. Citrus fruits, bananas, tomatoes, cabbage, and potatoes are valuable sources of vitamin C.

Lactating women vary in their requirement for vitamin D. In any case it is desirable to supply this vitamin in

addition to any small amounts that may be contained in the foods of the diet. At least 400 units daily should be given, and in some instances 800 units may be required for satisfactory calcium utilization.

When the diet contains the articles mentioned, it is rather immaterial what goes to make up the remainder; it is essential, however, that the total calories be sufficient to provide for the needs of the mother and for the secretion of milk as well. The nursing mother should take about 1,000 calories more than she would otherwise. She may make up the calories in almost any way she prefers, so long as the essential foods, milk, green vegetables, and fruits are taken in sufficient amount. She may take meat once a day or three times a day, she may take carbohydrate as potatoes, white bread, whole wheat bread, rye bread, or cereals, hot or cold. She may eat hot cakes and syrup and a reasonable amount of candy and cakes, provided her own digestion is not thereby upset.

A sufficient intake of fluids is essential. A nursing mother should drink at least a quart (liter) of water a day in addition to the quart and a half (1500 ml.) of milk. The water may be taken as fruit juices or other flavored beverages.

Mothers whose diets are insufficient in total amount to meet their own needs may secrete milk which is produced at the expense of the mother's body, but the amount secreted is likely to be scanty and to decrease gradually. Overstuffing the mother with food, however, will not increase her milk secretion. It is doubtful whether the proportions of fat, carbohydrate, and protein in the milk are greatly influenced by changes in the character of the mother's diet, although the vitamin content is susceptible to considerable change. No evidence has been presented that the taking of moderate amounts of alcoholic beverages or of tea and coffee or smoking in moderation affects the character of the milk adversely, provided the mother is not affected.

Relatively few drugs taken by mouth are excreted in the milk in sufficient amounts to have an effect on the infant. Atropine, morphine, iodides, bromides, arsenic, salicylic acid, nicotine, sulfonamides, and some of the coal tar antipyretics are secreted into the milk in small amounts. The taking of laxatives by the mother is generally believed to produce laxative effects on the nursed infant. This is difficult to explain, if true.

Care of the Breasts and Nipples

The breasts should not be compressed by close-fitting dresses or brassieres, but pendulous breasts should be loosely supported. Cleanliness of the breasts and nipples is essential. The nipples should be gently washed with clean (preferably boiled) water before and after each nursing. This should be done with clean cotton cloth or gauze. The nipples should not be handled with the fingers. A clean piece of cloth should cover the nipples between nursings.

If the nipples become tender or chapped, they should be greased with petrolatum, lanolin or 10 per cent balsam of Peru ointment.

The chief thing to be feared and avoided in connection with the mother's nipples is that they become chapped and fissured. Such fissures make nursing a very painful process and at the same time offer abundant opportunity for entry of pyogenic bacteria which may cause inflammation or abscess formation to such an extent as to make nursing not only temporarily, but even permanently, impossible. If the nipples have become fissured, it is well to allow the baby to suckle only through a nipple shield, keeping the breasts empty, if necessary, by manual expression or a breast pump. Often nipple shields prove relatively inefficient, in which case the baby should be fed milk that has been expressed from the breast. Healing of fissures is promoted by the application of a 5 per cent solution of silver nitrate. In the case of abscess formation, it is usually

necessary, at least temporarily, to wean the baby from the involved breast. The breast should be emptied by manual or mechanical means and the milk discarded until healing has occurred, when the nursing may be resumed.

When the milk secretion is first established, the breasts may become painful, due to engorgement. This same condition of engorged or "caked" breasts may occur from time to time subsequently. In such instances the milk flow may be diminished through the wearing of a tight binder and through restriction of fluids. At times it is necessary to relieve the engorgement by manual or mechanical expression of the milk. (For technique see p. 180.)

Technique of Breast Feeding

The baby should be placed at the breast for the first time six to twelve hours after birth, and thereafter every six hours during the next twenty-four hours. Very little colostrum may be obtained by the infant, but the nursing will tend to stimulate the flow of milk and will accustom the baby to suckling.

Beginning on the third day, the infant should suckle both breasts every four hours. Very little milk will be obtained during the first few days so that after each nursing the infant should be given sterile water, sugar solution, or a milk formula from a bottle. Sugar solutions, usually 5 per cent, are used extensively for this purpose. For those who prefer a milk formula, a suitable one may consist of one part of evaporated milk and two parts of water with 8 per cent added sugar. If by the fourth or fifth day the amount of milk obtained by the infant from the breast is insufficient, the complementary feedings of a cow's milk formula should be continued, or started if not given previously. Usually complementary feedings must be continued for only a few days. They should always be offered *after* the baby has been put to the breasts and has taken as much milk as he can obtain. A definite possibility exists that the newborn

baby will not suckle at the breast with sufficient vigor or persistence if he has become accustomed in the first few days of life to obtaining his food with greater ease from the bottle.

When the feeding interval is four hours, it is usually advantageous for the infant to suckle both breasts at each feeding. In this way more milk usually can be obtained than with three-hour nursings of alternate breasts. Occasionally when the milk secretion is scant, nursing both breasts at three-hour intervals is an effective means of increasing the flow. At times the infant is unable to empty the breast completely because of the presence of depressed nipples or because of weakness or mouth deformity. In such cases the breasts should be emptied mechanically and the expressed milk fed to the baby until the obtaining of a sufficient amount becomes possible.

A few infants fail to do well on a four-hour feeding schedule. Some become very hungry at the end of two and one-half or three hours, and cry until the feeding is given. This may be simply an indication of underfeeding. On the other hand, it may mean that the infant's gastric motility is rapid and his stomach is quickly emptied. Small or weak infants who do not suckle vigorously often do better on a three-hour schedule. If a three-hour feeding schedule is adopted, the breasts should usually be offered alternately except during the period when the milk flow is being established. It is not advisable to put the infant to the breast more often than every three hours.

The four-hour nursing interval has a number of advantages over shorter intervals. The infant is hungry at the end of four hours, suckles vigorously, and more completely empties the breasts. This in itself is the best stimulus to milk production. The gastrointestinal tract of the infant, especially the stomach, has a chance for rest between feedings and is not constantly taxed to capacity. The infant fed at four-hour intervals takes more food at a feeding and sleeps longer after the feeding, thus getting more rest

in the course of a day. Vomiting is less frequent in infants fed at longer intervals than in those fed at two- or three-hour intervals. The long feeding interval gives the mother a chance for rest and recreation and makes it unnecessary for her to spend her entire time with the baby. Some mothers, who are unwilling to nurse their babies at short intervals, do not object to the longer intervals.

The number of feedings in twenty-four hours will depend on the adequacy of the milk supply. When the mother has an abundant supply of milk, even a young infant may receive sufficient food in four or five nursings to meet all his requirements. As soon as the infant will sleep through the night without waking and is gaining weight at a satisfactory rate the 2 a.m. feeding may be omitted and later the one at 10 p.m. Occasionally, when the night feedings are omitted, the infants awaken at night, either through force of habit or because of thirst, and cry. The giving of a bottle of water is likely to satisfy them.

A feeding schedule once adopted should be adhered to within reasonable limits, but the baby should be allowed some latitude. Occasionally infants who are fed at four-hour intervals become very hungry at the end of three or three and one-half hours, especially when they have been active and when the previous feeding has been a small one. In these circumstances the infant should not be allowed to cry for a half-hour or an hour, but should be fed. The following feeding, however, should be at the regular hour. It is very easy for a mother to get in the habit of feeding the baby every time he cries and thus make the feeding interval shorter and shorter. This course of events is especially likely to happen in the case of babies who are definitely underfed because of a deficient milk supply. The amount of milk actually taken by the baby should be determined and if insufficient should be complemented by a cow's milk formula. Babies with irregular nursing habits are likely to be fussy and colicky, and the mother who feeds the baby whenever he cries becomes tired and nervous

and her milk supply consequently diminishes. An especially pernicious habit is that of having the baby sleep with the mother and suckle at short intervals during the night. The baby should be in a crib by himself, preferably in another room.

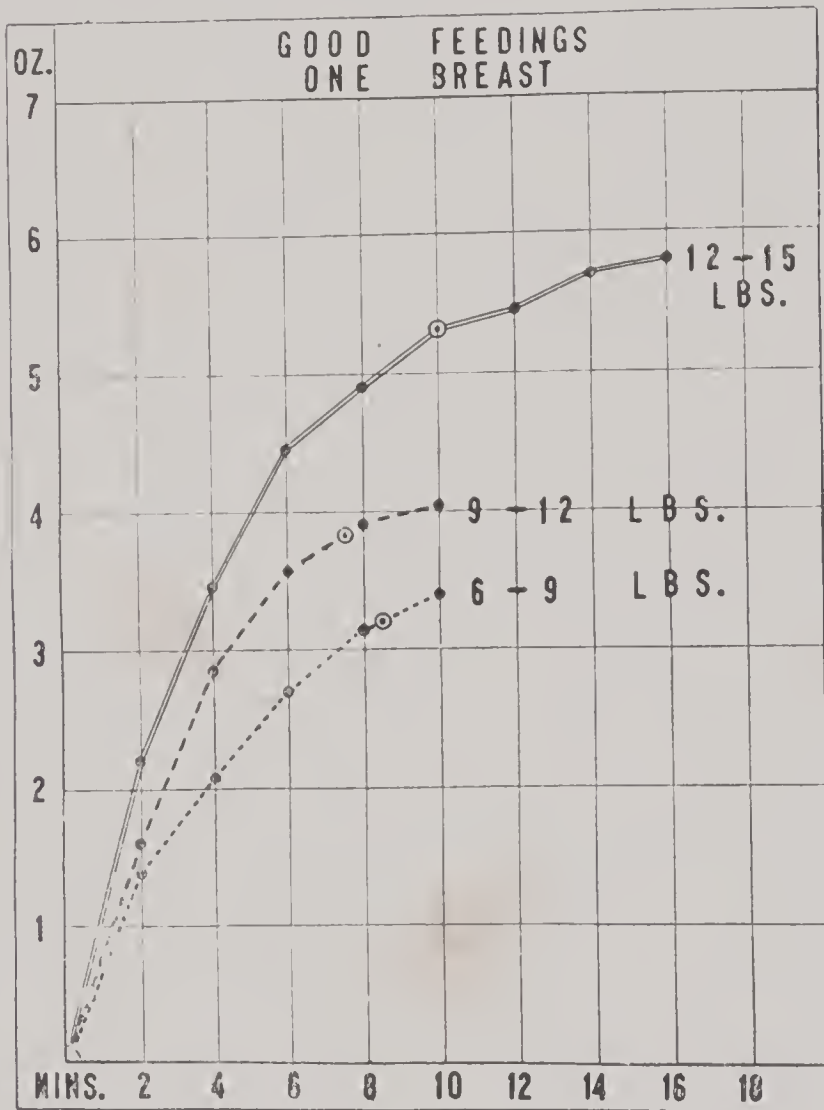


Fig. 7.—Curves showing rate at which milk is taken at a nursing by normal infants of varying weights. Average time after which no more milk is obtained is shown by circle surrounding dot. (Smith, C. H., and Merritt, K. K., *Am. J. Dis. Child.* xxiv, 413, Nov., 1922.)

The number of minutes that an infant feeds depends on the sucking strength of the infant, the amount of milk available, and whether the breasts are difficult or easy to empty. Under normal conditions, a vigorous infant will obtain as much as one-half of the milk in the first two or

three minutes and three-fourths of the total within the first five minutes. Very little milk is obtained after ten minutes. Small and weak infants obtain milk at a considerably slower rate. These facts are well illustrated in the accompanying chart of Smith and Merritt (Fig. 7).

A healthy baby feeding at both breasts should not suckle longer than ten minutes at each, and one feeding at a single breast, not longer than fifteen minutes. Some very weak infants may feed as long as twenty minutes. Infants who remain at the breast for long periods are likely to swallow air instead of milk, and this tends to cause vomiting and possibly colic.

The position of the baby during the nursing is of importance. A few mothers prefer to nurse their babies while lying down. It is better that the mother sit up while nursing the baby and that the baby be held in a semierect posture; the swallowing of air is then largely avoided and the air already in the stomach tends to pass up the esophagus as the milk is swallowed. It has been shown that infants held in a semierect posture usually take more milk at a feeding than those who are fed while recumbent. This is due to the fact that the intake of food is not interfered with by distention of the stomach with air. All babies swallow a certain amount of air, and roentgenograms regularly show an air bubble in the stomach. There may be sufficient air almost to fill the stomach. In all instances, before the nursing the baby should be held upright over the mother's shoulder and patted on the back until any swallowed air is belched up. The infant should also be held up immediately after the nursing. With some infants, it is necessary to interrupt the nursing to allow swallowed air to escape.

The amount of milk obtained at a single nursing is not uniform and depends upon the appetite of the infant and the amount of milk secreted by the mother. A healthy normal infant may take eight ounces at one feeding and three at another. If an infant has slept for three or four hours

between feedings, he is likely to take less than if he has been awake and active. After a long night interval a much larger amount of milk is likely to be taken than at other feedings during the day. It is consequently impossible to state even approximately the volume of milk a breast-fed baby should be expected to take at a single feeding, although the total amount required for each day may be stated more definitely. No idea as to the adequacy of the milk intake can be obtained from an observed single feeding. To determine the total daily amount of milk received by an infant, he should be weighed before and after several feedings on successive days and the results averaged. Weighings should be performed with clothes and diapers on, for if the diaper is changed the loss of weight due to passage of urine or stool may lead to erroneous conclusions as to the intake of milk.

The amount of milk received by the breast-fed baby increases after birth until at two weeks of age he gets an average of 130 grams for each kilogram (2 ounces for each pound). By two months of age he should be getting at least 150 grams for each kilogram ($2\frac{1}{3}$ ounces for each pound). With an energy value of 665 calories to the liter (20 calories to the ounce) the milk received by the baby at two months will supply at least 100 calories for each kilogram (45 calories for each pound). If an infant fails to gain, is fretful and hungry, and it is found that he is receiving an amount of milk less than the quantity stated, it may be assumed reasonably that the food is inadequate. If, on the other hand, an infant fails to do well despite receiving as much as 165 grams for each kilogram ($2\frac{1}{2}$ ounces for each pound), the probability is that the trouble is not due to underfeeding.

Characteristics of the Normal Breast-Fed Infant

The normal, breast-fed infant is well nourished, his flesh is hard and firm, his eyes are bright, and his cheeks and nails are pink. He is happy and active when awake. He

cries only when hungry or uncomfortable from such causes as cold, a wet diaper, improperly adjusted clothing, or distention of the stomach from swallowed air. He has from two to four stools daily during the first few months and from one to three daily after that time. The normal breast-fed baby gains from 180 to 240 grams (six to eight ounces) a week throughout the first year (see weight curves, Chapter I). A healthy breast-fed baby usually sleeps from fourteen to eighteen hours a day during the first two months, and from fourteen to sixteen hours a day during the remainder of the first year.

Infants nursed at the breast usually do well provided the mother has a sufficient supply of milk and the baby is fed at proper intervals. Failure to do well is more likely to be due to the presence of infections or to congenital anomalies than to the character of the food if the quantity of food is satisfactory.

Underfeeding

Certain breast-fed infants fail to receive a sufficient amount of milk from the mother or spit up frequently and are consequently underfed. The symptoms of underfeeding depend on the degree of deficiency of the diet. There may be only a small gain in weight, or an actual loss. The total volume of stools passed in a day may be small, but the individual movements may be frequent and green. The diarrhea of starvation may be mistaken for that of overfeeding. The underfed infant is fretful, cries a great deal, and is likely to suffer from colic. The underfed baby sucks his fingers and anything he can get to his mouth and consequently swallows a great deal of air and often vomits from this cause. Hunger causes painful contractions of the stomach and gastrointestinal tract. If the total amount of milk obtained by the baby is found to be small, complementary feedings from the bottle should be given. At the same time, every effort should be made to improve the mother's physical condition and her diet, if inadequate,

should be corrected. This phase of the subject has been discussed previously in this chapter.

There are no drugs which regularly increase the flow of milk. A temporary increase in the milk occurs when pituitrin is administered, but this appears to be due merely to the forcing out of milk from the breasts. That fraction of pituitary extract known as prolactin has been used for increasing the amount of milk.

If, despite all efforts, the amount of milk obtained by the baby is insufficient for his needs, the complementary cow's milk formula is continued, or else the baby is put to the breast for only a few feedings a day and a bottle is given at the remaining feedings. It is usually inadvisable to wean the baby completely during the first few months of life if the mother is able to give as much as one-half of the milk necessary for his nutrition. When no more than this can be obtained, even after making every effort, it is not worth while to continue the breast feeding and the infant should be weaned completely.

Overfeeding

If an infant is nursed at intervals of four hours, there is little danger of overfeeding at the breast. Occasionally the mother has an abundant supply of milk which flows so freely that the infant gorges himself at each feeding and promptly regurgitates at the conclusion of the nursing. This does relatively little harm, the spitting up of the food protecting the infant's intestinal tract from too great an excess. Sometimes the infant may retain more milk than his digestion is capable of caring for, and indigestion or diarrhea may result. The simple remedy is to limit the time of nursing, allowing the infant to suckle only one breast at a feeding and then for a short period, which may have to be only three or four minutes. If the baby is overfed when being nursed at three-hour intervals, a four-hour schedule should be employed.

Unsuitable Milk

Milk from a healthy and suitably fed mother rarely disagrees with her baby, although the baby may fail to do well because of irregular nursings or ingestion of insufficient quantity of milk. In those instances in which the milk seems to disagree, the cause usually is not easily found. When the quantity of milk is sufficient, analysis of the milk for fat, protein, and carbohydrate seldom gives helpful information.

The amount of fat in human milk varies widely among different women. The fat content may be low so that the milk has a low energy value or the fat content may be so high as to lead to digestive disturbance. In some instances in which the fat content is high, the difficulty may be remedied by having the infant only partially empty the breasts at a nursing. This practice, however, is likely to result in decrease in milk secretion.

In those instances in which milk of average composition disagrees with the baby, the possibility of allergy is to be considered. Cases have been reported in which the infant appeared to be sensitive to foreign proteins in the mother's milk. Elimination of the suspected proteins from the mother's diet is indicated in such cases. Eggs appear to be a cause of sensitization more frequently than other foods.

Infants may fail to thrive because of vitamin deficiency in the mother's milk. The vitamin content of the milk can best be improved by giving the mother a good diet.

When breast-fed babies fail to do well despite regular nursing, sufficient food intake, proper care, absence of infection, and a good maternal diet, weaning may be necessary.

In a commendable effort to encourage breast feeding many extreme statements have been made, such as "a mother's milk always agrees with her baby," "a baby who

does not do well at the breast will do worse on artificial feeding." Such statements are untrue. All human milk is not perfect any more than all human beings are perfect.

Gastrointestinal Disturbances of Breast-Fed Infants

Severe gastrointestinal disturbances occur rather infrequently in the case of infants who are exclusively breast fed. Minor disturbances such as colic, spitting up of food, constipation, or mild diarrhea are, however, of fairly frequent occurrence and call for appropriate treatment. Each of these conditions is considered in detail in individual chapters that follow.

Mixed Feedings

There are a number of conditions under which it is advisable to feed the infant partially from the breast and partially from the bottle. This may be necessary during the time that the milk secretion is being established. It also becomes necessary when the milk supply begins to diminish or becomes insufficient for the increasing needs of the growing infant. Weaning is usually accomplished by the gradual substitution of the bottle for the breast. Mixed feeding may be carried out in two ways, the artificial formula being given either as *complemental* or as *supplemental* feeding. By complemental feeding is meant the giving of a bottle to complete a single feeding. The bottle feeding in such instances should always be given after the infant has been fed at the breast. By supplemental or substitute feeding is meant substitution of the bottle for one or more of the breast feedings during the day.

Complemental feedings are usually used when the mother's milk is insufficient for the infant's needs and an attempt is still being made to increase the amount. Supplemental feedings are used at the time of weaning when it is desired to allow the breast to dry up gradually. Supplemental feedings are also used occasionally in the case of

normal infants whose mothers have sufficient milk but are required to be away from home daily for a longer time than the customary feeding interval. The giving of one bottle a day even when not necessary is occasionally advised because of the fact that this accustoms the baby to the artificial feeding so that no difficulty is likely to be experienced if the baby has to be weaned suddenly or the mother has to miss one or two nursings. The disadvantages of this practice are those of artificial feeding in general. The milk mixture may not be suitable for the baby or it may be contaminated by bacteria due to lack of care in its preparation. A further disadvantage is that the missing of one or two nursings a day tends to diminish the mother's milk supply.

In the giving of a mixed feeding, the cow's milk formula should be one suitable in amount and composition for the age and size of the infant. The formula will be approximately the same as that used in the case of infants of the same age who are entirely artificially fed. The construction of these formulas is considered in the chapter on artificial feeding. Very young infants may be given either sweet milk or acid milk. Older infants, however, will often refuse to take the bottle if it contains acidified milk because of the great difference in taste as compared with human milk. Sweet-milk formulas usually must be used. Sucrose, on account of its greater sweetness, may be the preferable carbohydrate to add to the formula for mixed feedings.

Additions to the Diet of the Breast-Fed Infant

It is advisable to give cod-liver oil or other suitable source of vitamin D to the breast-fed infant. It is particularly necessary in the case of prematurely born infants, those growing very rapidly, and infants of certain dark-skinned races. Cod-liver oil is not so necessary if the infant is frequently exposed to sunlight but should be given in all instances during the cool months. The administration of cod-liver oil should be begun early in the first month. One teaspoonful daily (300 to 400 units of vitamin

D) is sufficient. This may be given at the time of nursing, the oil being fed with a dropper, or it may be fed by spoon at the same time that the orange juice is given.

It seems desirable to give orange juice, or some supplement equivalent in vitamin-C content, to the breast-fed baby even though the milk of the well-fed mother contains sufficient vitamin C. The average observed content of vitamin C in human milk is much lower than the maximum values reported. Because of the amount of the vitamin in human milk, the need of the breast-fed baby for additional vitamin C is not so great as in the case of the artificially fed baby, but an additional amount is not harmful and it serves as a factor of safety. Also the feeding of orange juice increases the variety of flavors to which the baby should become accustomed early. One ounce of orange juice should be given daily during the first three months, and two ounces thereafter.

Other additions to the diet become necessary as the age of the baby increases. The needs of the breast-fed baby in respect to these additions are essentially the same as those of the artificially fed infant. Giving the breast-fed baby soft custard at three to four months of age is desirable. This addition increases the protein, mineral, and vitamin content of the diet appreciably. Such a procedure also accustoms the baby to spoon feeding early, so that final weaning to cup and spoon will be simplified. In order to avoid repetition, the feeding of egg, fruits, vegetables, cereals, and meat products is discussed in Chapter XVIII.

Weaning

Under ordinary conditions, weaning should be begun about the eighth or ninth month. Frequently, however, insufficiency of the mother's milk may make it necessary to wean the baby partially or completely before this age. It is possible for babies to be breast fed successfully beyond the tenth month if the diet is appropriately supplemented

with fruits, vegetables, and other foods, but no particular advantage is gained by so doing. Whether or not babies should be weaned electively during hot weather depends much on the socioeconomic status of the parents and on related factors. When good milk refrigeration and general sanitary care are possible, there can be no important objection to summer weaning appropriately managed. The greater likelihood of bacterial overgrowth in the milk in the summer and of decreased digestive capacity on the part of the infant during hot weather must be kept in mind. The weaning of a baby at an early age in the spring in order to avoid weaning at an appropriate age in the summer does not seem justifiable, although often recommended. If possible, weaning should be avoided while the infant is acutely ill.

When weaning becomes necessary before the sixth or seventh month, it is usually desirable to wean to bottle feeding. When babies are weaned at seven months or later, it is as easy to wean to cup and spoon feeding as to bottle; such a procedure avoids the necessity and possible difficulty of weaning from the bottle when the baby is a few months older. Unless the infant has been accustomed to taking food from the bottle or cup, weaning is sometimes difficult because of obstinate refusal to accept a food source other than the breast. No difficulty is to be expected if the baby has been fed previously from the bottle or cup and if these experiences have proved satisfying. This type of preparation should always be given. When sudden weaning becomes necessary without previous preparation, the food should be offered at regular intervals. Usually the baby will yield. Occasionally gavage is necessary.

Wet Nursing

In a previous generation the mortality of infants deprived of mother's milk was tremendous and about the only hope of rearing such an infant successfully lay in procuring milk from another woman. Formerly also it

was the custom to feed the foster baby directly at the breast of the wet nurse. At the present time most infants deprived of mother's milk are fed artificially with such success that wet nursing is now rare. It is chiefly for prematurely born and ill infants that human milk remains desirable at times. In these circumstances the infants are fed milk that has been expressed from the breast. Milk from foster mothers is a makeshift substitute for maternal nursing and is commonly used only for an emergency period.

The method of obtaining human milk varies with local conditions. In hospitals with a large maternity service milk may be obtained from those mothers under care who have an excess beyond the needs of their own babies. In some cities "breast milk dairies" have been established to serve as distributing centers for human milk. The wet nurse expresses her milk and brings it to the center. When neither of these facilities is available and when the need is sufficient, usually a mother can be found who has milk to spare and the milk can be delivered to the home of the foster baby. Many healthy lactating women are able to supply sufficient milk for two or three infants.

In selecting women for the purpose of supplying human milk no attention need be paid to the age of their infants, as the milk does not change greatly in composition after the first month until the end of the lactation period. The woman should be in good health. She should be free from tuberculosis and syphilis. She should be cleanly in her habits and her own baby should be thriving, this being a good indication of the quality of her milk. It is immaterial whether she be white or black, as biological characteristics are not transmitted by way of the milk. Colored women are more likely to have an abundant supply of milk than white. On the other hand, they are more likely to have poor diets so that the milk may be deficient in quality.

The wet nurse needs careful training in methods of collecting milk without contamination. When collected for distributing centers, the centers provide sterile containers; the collected milk is pooled, sterilized, and desiccated or frozen to preserve it.

Manual or Mechanical Expression of Milk from the Breast

A number of conditions occur in which it is necessary or desirable to express the milk from the breasts. Complete emptying of the breasts is the best stimulus to milk secretion, and when the infant is unable to drain the breasts because of weakness or mechanical defects, forcible expression of the milk often serves to maintain or to increase the amount of secretion. It is particularly during the early weeks of the infant's life that mechanical expression of the milk is indicated.

When the nipples are fissured or infected the infant should not be nursed, but the milk should be drawn off. Mechanical emptying of the breasts is also sometimes indicated in the case of engorged or caked breasts. The milk of a wet nurse is usually expressed for the purpose of feeding infants.

Milk may be expressed manually or by the use of a variety of mechanical appliances. Manual expression, when properly performed, is very effective, and the technique is not difficult. The principle of manual expression is to empty the milk sacs which hold the accumulated secretion. These sacs lie *behind* the nipples so that mere pulling, squeezing, or stripping of the nipple is not sufficient. The proper technique is for the mother to grasp the breast about an inch and a half from the nipple with the fingers below and the thumb above. Firm pressure is then made backward against the breast and with the hand in this position the fingers and thumb are brought together forcibly. This motion should be repeated from 60 to 100 times a minute. The fingers should not be allowed to slip forward on the skin. The milk is caught in a sterilized glass,

which is held in the other hand. The glass need not touch the breast, as the milk is forced out in streams to a considerable distance. A mother who is skillful in the manual expression of milk can often remove a considerable amount of milk from the breast after the infant has obtained all that he can.

Breast pumps are of several varieties. The common glass type with collapsible rubber bulb is not very efficient and its use is often painful. Electrical or water pressure pumps are, however, very effective. The electrical breast pump has found wide use, especially in hospital practice. The principle is that of an intermittent sucking motion, the funnel placed at the breast being of such shape that compression of the milk sacs occurs coincident with the suction.

An effective and inexpensive pump is one that uses water power for suction. A water suction pump is attached to any convenient faucet. The receptacle for milk collection is equipped with a three-hole stopper with appropriate tubing. One tube connects with the water pump, another with the funnel placed at the breast and the third is used to make the pressure intermittent by closing and opening at appropriate intervals.

CHAPTER XII

ARTIFICIAL FEEDING

General Considerations

When an infant is deprived of human milk, the natural substitute is milk from some other species. Cow's milk is usually employed in this country, but the milk of goats or other animals is occasionally used. Also, infants may be fed successfully with mixtures containing no milk.

The milk of each species differs from that of others in relative amounts of organic and mineral constituents, caloric value, physical properties, and degree of digestibility. If one has a knowledge of the nutritional requirements and the digestive capacity of the infant, it is possible to prepare feedings from cow's milk or from the milk of some other species which meet all of the requirements and which are within the limits of capacity of the infant's digestion. It is also possible to render the feedings free from harmful bacteria by sterilization. In the preparation of artificial feedings, the chance exists that some essential constituent of the diet may not be present in adequate amount or that bacterial contamination may occur after the feedings have been prepared.

The earliest attempts at infant feeding consisted merely in the giving of cow's milk or the milk of some other animal in such amounts as the infant would take. It was found that some infants could be reared successfully in this way, but many succumbed to gastrointestinal infections or nutritional disturbances. Most of the high mortality among artificially fed infants in the past has undoubtedly been due to bacterial contamination of the food. Before the advent of modern methods of milk production and preservation, much of the milk obtainable was badly contaminated, especially during the warmer seasons of the year. The

importance of bacterial contamination of milk has been appreciated only comparatively recently.

Failure of infants to do well when artificially fed was originally believed to be due primarily to differences in the composition of cow's milk as compared with human milk which rendered it indigestible and otherwise unsuitable for the needs of the infant. Attempts at the modification of milk by dilution or the addition of various substances met with relatively little success until the factor of bacterial contamination was considered and methods were adopted to insure the absence of harmful bacteria. It was found when infants were fed milk obtained under sanitary precautions and used fresh, that good results were obtained in a much larger proportion of cases than when unclean and carelessly handled milk was used. Subsequently it was found that boiled milk was better tolerated even than clean raw milk, but that many infants fed clean, boiled cow's milk without other modification did not thrive so well as those fed human milk.

Many attempts have been made to explain the causes of failure in the case of artificially fed infants. Almost every constituent of cow's milk has been incriminated as the causative factor in explaining this difference. The underlying idea of the earlier methods of modifying milk for infant feeding was that some constituent was harmful and must be reduced in amount or removed. This concept culminated in the comprehensive system of Rotch, usually referred to as the "percentage method" of feeding. The theoretical basis of this system was that the nutritional disturbances of artificially fed infants resulted from the harmful effects of either fat, carbohydrate, or protein; that an excess of any one of these components caused characteristic disturbances; and that the remedy lay in the reduction of the harmful component or components in the diet. Reduction of protein was effected by dilution of the milk, the fat and sugar contents being restored by additions of cream and milk sugar. The appearance of certain symptoms called

for reduction of either the sugar or fat, in addition to reduction of protein. Fat alone was reduced by removal of the top portion of the milk. In intelligent hands such methods of modification of milk have led to successful feeding results, but all too often misinterpretation of symptoms has resulted in a reduction of the food constituents to a point below the nutritional needs of the infant, and as a consequence infants have been fed digestible but insufficient food.

It has long been known that the curds produced by the action of acid or rennin on cow's milk are larger and tougher than those from human milk and this is generally believed to be one of the factors in rendering cow's milk less digestible. Dilution of the milk with water leads to smaller curd formation. Dilution with cereal gruels or lime water is more effective still in reducing the size of the curds. Heating of the milk to high temperatures changes the physical properties of the casein so that the curds produced are smaller.

Certain other physical properties of cow's milk also have been implicated as factors in rendering it less readily digestible. Although cow's milk normally is very slightly more acid than human milk, much larger amounts of acid are required to bring it to the same degree of acidity as is attained by human milk during gastric digestion. This high "buffer value" has been considered to have a retarding effect on gastric digestion through excessive neutralization of gastric acid. In order to overcome this effect, milk is often modified by the addition of various acids. Appropriate acidification prevents the formation of large curds in the stomach and it is probable that this factor is more important to the infant than is reduction of the buffer value.

In contradistinction to the older theories that cow's milk contains an excess of some harmful substance, there is the

more recent viewpoint that cow's milk is deficient in certain essential constituents, such as iron and certain vitamins.

Without question, all of the theories which have been advanced to explain the relative unsuitability of cow's milk for the feeding of the human infant contain certain elements of truth, and every attempt at modification based upon the various theories has served to increase our knowledge of the nutritional needs and digestive functions of the infant and has brought us nearer to the solution of the problem of satisfactory artificial feeding. There is no one method of artificial feeding which is the only correct one. Infants may be fed artificially in a variety of ways and good results obtained. On the other hand, any system, unintelligently used, results in numerous failures. No matter what method is used for the preparation of the formulas, certain essential requirements must be fulfilled.

The Requirements of a Satisfactory Artificial Feeding

The nutritional requirements of the infant are fairly definitely known and the capacity of the infant to digest the various food components either singly or in combination is also known. The effects of bacteria introduced by way of the milk are understood, as are the methods for rendering the milk free from harmful bacteria.

With our present knowledge it is possible to formulate the essential requirements of satisfactory artificial feeding, as follows:

1. Sufficient calories.
2. Sufficient protein, carbohydrate, fat, mineral salts, water, and vitamins.
3. Absence of harmful bacteria.
4. Easy digestibility.

Any form of food that meets the above requirements will be successful, whether it be prepared from cow's milk or human milk, whether it be prepared from fresh liquid

milk or from evaporated or dried milk, whether it be sweet or sour, and whether or not there be any milk at all present. It will make little difference whether the basis of the diet be a proprietary food obtained from the drugstore or simply milk and sugar obtained from the grocery store. Good results may be obtained provided only that all of the above requirements are met. Failure to meet any one of these requirements will result in failure of the feeding as a whole.

Only too often, a food which is easily digestible is so dilute or given in such small quantities that insufficient calories are provided. Attempts to render the feeding digestible may result in the giving of too little protein, with consequent small gain in weight and poor nutritional results. This is the common fault of many of the high-carbohydrate proprietary foods which contain relatively little milk. A formula suitable in respect to all food constituents may be contaminated by harmful bacteria. In the effort to meet all the requirements, a food may be prepared which is quite beyond the capacity of digestion of the infant. So much attention, however, has been centered on the digestibility of the food that this error is not one which is frequently made.

If one keeps the essential requirements clearly in mind, it is not difficult to prepare a satisfactory artificial feeding adapted to the needs of the individual infant. A clear understanding of these fundamental principles is also of the greatest aid in determining the causes of unsuccessful feeding. A review of the history of "difficult feeding cases" usually reveals the fact that previous feedings have failed to meet one or more of the essential requirements. Once having determined the essential error or errors, correction of the discrepancy may be expected to remedy the feeding difficulties. It should, however, be pointed out that improper feeding is only one of the causes of the failure of infants to thrive. Only too often blind efforts are made to find a suitable formula for an infant who is not thriving when the real difficulty is organic disease or undiscovered

infection. If the feeding is one which meets all the requirements, it may be assumed confidently that some other factor is at fault and a careful search should be made to determine this disturbing factor.

The requirements as to calories, protein, carbohydrate, fat, mineral salts, water, and vitamins have been considered in detail in Chapters II to VII and are summarized in Chapter VIII.

Bacterial Contamination of the Infant's Food

Harmful bacterial contamination of milk may be prevented by scrupulous care in production, distribution and handling. Certified milk is the cleanest form of raw milk available and is usually free from harmful bacterial contamination. It is, however, safer in all instances to subject milk or milk formulas used for infant feeding to a sufficient degree of heat to insure destruction of any pathogenic organisms. Pasteurization of milk, when properly carried out, is an effective method of rendering it safe from disease-producing bacteria. The custom of boiling all milk for infant feeding not only makes it bacteriologically safe, but alters favorably its buffer value and curd properties. Evaporated milk is sterile and dried milk is usually, though not always, free from harmful organisms. The prepared baby foods contain varying numbers of organisms, depending upon the care taken in the processes of manufacture. Acidification of milk mixtures to a sufficient degree results in inhibition of bacterial growth and tends to prevent further growth after formulas have been prepared.

For a more detailed discussion of the bacteria of milk and of the methods of reducing bacterial contamination, see Chapter XIII.

Milk is not the only source of bacterial contamination of the infant's food. Contamination may occur from the use of impure water, from lack of cleanliness of bottles and nipples, or from accessory foods.

Chemical Contamination of the Formula Diluent (Well Water Cyanosis)

City water supplies may be expected to be safe for infant feeding. Much more often than is realized rural wells are contaminated by surface drainage. The boiling of such water should make it bacteriologically safe for infants. However, the contaminated water is very likely to contain nitrates in quantities detrimental to young infants. The nitrates of the water are changed to nitrites by bacteria in the intestinal tract. Nitrites, when absorbed, change hemoglobin to methemoglobin, a change that results in cyanosis. The greater the proportion of water in the formula, the greater the cyanosis. Thus, formulas prepared from evaporated milk are more likely to cause cyanosis than those prepared from fresh milk because of the greater dilution. Blood drawn during a period of cyanosis has a peculiar chocolate color that is distinctive. Babies recover spontaneously within a day or two after use of the contaminated water is stopped. When the cyanosis is severe and alarming, it is desirable to cause recovery more quickly. Dramatic return to normal skin color and appearance of the infant can be produced by intravenous injection of 0.15 ml. for each kilogram (0.07 ml. for each pound) of a 1 per cent solution of methylene blue.

Digestibility of Milk Formulas

As discussed elsewhere (Chapters IX, XI, and XIII) raw cow's milk is less readily digested by the infant than is human milk. Cow's milk that has been heat-treated is more readily digestible than raw milk. Heat treatment of milk alters both the buffer value and the size of the casein curd resulting from the action of acid and rennin in the stomach. Both of these changes are in the direction of making the milk more quickly digestible. Boiled, evaporated, and dried milks have a lowered buffer value and tend to produce small curds in the stomach, which are passed from the stomach more quickly and acted on by the diges-

tive juices more readily than the coarser curds from raw milk. Milk appropriately acidified is more readily digestible for the same reasons—the smaller curd and the lowered buffer value. Similarly, diluted milk is more quickly digested than whole milk. The common procedure of moderately diluting boiled milk for feeding the young infant results in a formula readily digestible by all healthy infants.

It seems desirable to make a distinction between speed of digestion and ease of digestion. The various modifications of milk discussed cause increased speed of digestion, but only in the most limited sense do they increase the ease of digestion. Except for the passage of casein curds in the stools, never encountered with milk heated to boiling temperature, protein is completely digested before the lower colon is reached, regardless of the method of modification. The methods of modification discussed, except dilution, affect only the casein as far as digestion is concerned. Thus we are not concerned with incompleteness of protein digestion, but rather with the speed with which the stomach empties and with the orderliness of intestinal digestion. Food which remains in the digestive tract of the infant when digestion, for any reason, is proceeding with unusual slowness is subject to excessive bacterial decomposition—a condition that readily leads to “indigestion” with gastrointestinal symptoms.

It is not possible to state precisely how much cow's milk the infant can digest without symptoms of indigestion, nor is it possible to state accurately the relative digestibility of milk modified in the various customary manners. From clinical and laboratory investigation, however, it may be stated in very general terms that boiled whole milk requires approximately two or three times as long to digest as human milk, and milk diluted to one-third or one-half is about as quickly digested as human milk. Acidified whole milk approximates human milk in speed of digestion. The

same may be said of reconstituted evaporated and dried milks, particularly if appropriately acidified.

The capacity of the infant to digest cow's milk without symptoms of indigestion varies greatly with age and physical condition, as well as with the character of the previous feedings. Infants who have been fed cow's milk from an early age become accustomed to it and are able to digest more than infants who previously have received only human milk. The greater the dilution of boiled whole milk, the more readily is it digested. However, extreme dilution not only is unnecessary but does not allow an adequate food intake. By four months of age a baby usually will take milk diluted only one-third or one-fourth, and by seven or eight months most babies can take undiluted boiled whole milk. Acidified milk can be given undiluted, if desired, from the beginning, as may also other modifications with similar properties. Skimmed or partially skimmed milk may be expected to leave the stomach more quickly than whole milk.

Preference of the Infant for Various Types of Formulas

Clara Davis has studied the preferences among infants for feeding mixtures. Infants in institutions were offered in rotation at each feeding sweet milk feedings of two dilutions and acid milk feedings. The order of rotation was changed at each feeding. The amounts of each type of food ingested were recorded and the data analyzed. The infants less than one month of age preferred the sweet milk feedings, older infants, the acid milk feedings. The dilute feedings apparently were the least liked.

CHAPTER XIII

COMPOSITION AND CHARACTER OF COW'S MILK

All cow's milk is not the same. The chief variations are in the fat content and in the number and character of contaminating microorganisms. The milk from Holstein cattle is somewhat more dilute than the milk of such breeds as Jersey and Guernsey. The high fat content of the latter is somewhat excessive for infant feeding. Some individual variation exists in the milk of cows of the same breed and of the same cow at different times. The mixed milk from a herd is far more uniform in composition than the milk from a single cow. Furthermore, the effect of any deleterious changes in the milk of any one cow in the herd is minimized by dilution of this milk with that of the remainder of the herd. Milk which is distributed by large dairies in cities is usually more uniform than country milk or milk from small dairies because of the fact that it is "standardized" to a definite fat content, which is often just a trifle above the minimum legal requirement. The amounts of the other components of milk, the protein, sugar, and mineral salts, are remarkably constant. The average percentage composition of Holstein and Jersey milks is

	Holstein	Jersey
Fat	3.5	5.0
Lactose	4.6	4.9
Protein	3.2	3.8
Ash	0.7	0.8
Water	88.1	85.4

Details as to composition of average milk are given in Table IV, page 158. The average energy value of cow's milk is 20 calories to the ounce, or 670 calories to the liter. Holstein milk, often used in infant feeding, has an energy

value of approximately 18.5 calories to the ounce, or 630 calories to the liter.

The chemical composition of the fat of cow's milk differs from that of human milk in that it contains a larger proportion of the glycerides of the volatile fatty acids and less linoleic acid which is essential for many species.

In general, the fat of cow's milk is somewhat less completely absorbed from the gastrointestinal tract and is somewhat more likely to produce gastrointestinal irritation than an equal amount of the fat of human milk. (See Chapter V for further discussion.)

The sugar of cow's milk is identical chemically with the lactose of human milk, although the total amount present is less.

The protein of cow's milk is mostly (85 per cent) casein, the remainder being whey protein (lactalbumin and lactoglobulin). The characteristics of these two proteins have been considered elsewhere. (See Chapters III, IX, and XII.)

The mineral constituents of cow's milk are more abundant than those of human milk and exist in different proportions. Cow's milk contains more calcium, magnesium, and phosphorus and less iron than human milk. (See Table IV, p. 158. Also Chapter VI.)

The vitamin content of cow's milk is discussed in detail in Chapter VII and summarized in Table IV, page 158. Milk contains sufficient vitamin A and riboflavin to meet the requirements of the infant. The amount of thiamine present in fresh milk or in milk quickly boiled and cooled is usually adequate to meet minimum needs. Prolonged heat treatment of milk is destructive to thiamine, and milk so treated may contain only sufficient thiamine to prevent clinical evidence of deficiency. Milk is a poor source of nicotinic acid, but obviously it contains enough to prevent pellagra. The amount of ascorbic acid in pasteurized or quickly boiled milk may be expected to be sufficient to prevent scurvy, but only just enough, and the amount

present under the best conditions is less than is considered desirable for optimum nutrition. The amount of vitamin D in unfortified milk is so small as to be practically negligible in relation to the infant's needs.

The buffer value of cow's milk, that is, the capacity to absorb considerable quantities of acid and alkali without significant change in chemical reaction, is much higher than that of human milk. It is necessary to add approximately three times as much acid to cow's milk in order to bring it to the optimum point for gastric digestion as in the case of human milk.

The Enzymes of Milk

Raw cow's milk always contains a number of enzymes of various types. Some of these are proteolytic, resembling either trypsin or pepsin in their action. Carbohydrate-splitting and fat-splitting enzymes also are present as well as oxidizing and reducing enzymes. There is a question as to whether these enzymes are natural, normal constituents of the milk or whether they are derived from the bacteria present. The enzymes in question exist in too small quantities to be of any practical significance in the subsequent digestion of milk by the infant. They are all destroyed by heating and this destruction does not make the milk any less digestible.

Miscellaneous Constituents of Milk

Cow's milk occasionally contains substances capable of causing disturbances in those drinking the milk. When cows are first turned out to pasture in the spring, and overeat, the milk occasionally causes gastrointestinal disturbances in infants. Certain poisonous plants may be eaten by cows, the most dangerous of which is probably snakeroot. The harmful substance is secreted into the milk and gives rise to the disease known as "milk sickness." Epidemics of milk sickness are mostly seen toward the end of a dry summer when the grazing has become poor and cows ingest plants which ordinarily they would not eat.

Bacteriology of Cow's Milk

The bacteria of cow's milk vary in character and number, depending upon the presence or absence of mastitis and the conditions under which the milk is produced and kept before use. Freshly collected milk contains relatively few bacteria (100 to 1000 to the cubic centimeter). Milk, however, is an excellent culture medium for most bacteria so that such microorganisms as may be accidentally introduced multiply rapidly unless the milk is kept at a low temperature. Milk is easily contaminated by stable dirt, unclean utensils, or the hands of milkers.

When milk is produced under clean conditions, kept at temperatures below 50° F. during transportation, and delivered to the consumer without unnecessary delay, the bacterial count ordinarily varies from 10,000 to 50,000 to the cubic centimeter. Properly sanitized milk having a bacterial count of less than 50,000 is ordinarily considered of good quality. Milk which has not been obtained under clean conditions, or which has not been cared for properly, may have a bacterial count of several million on delivery. The bacterial count of milk serves as an indication of the care taken in its production. Milk with a high bacterial count is not necessarily harmful to health as the bacterial flora may be entirely innocuous. On the other hand, milk may have a low bacterial count, but contain pathogenic organisms and therefore be unsuitable. In general, milk with a high bacterial count is likely to contain numerous types of organisms, some of which may lead to serious digestive disturbances. It has been shown by actual statistics that infants fed market milk of low bacterial counts are less subject to gastrointestinal diseases than those fed milk with high bacterial counts.

In general, the bacteria of milk fall into three main groups:

1. **Harmless, Lactic Acid Producing Organisms.**—These, under ordinary conditions, make up well over 90 per cent

of the total bacterial flora of raw milk. Included in this group are the *Lactobacillus acidophilus*, *Lactobacillus bulgaricus*, *Streptococcus lactis*, *Streptococcus faecalis*, and *Aerobacter aerogenes*. Strains of the colon bacillus of bovine origin, growing in a medium rich in sugar, ordinarily produce only lactic acid and other harmless products; other strains, however, growing under different conditions, may produce harmful products. The harmless acid producers, when growing in milk, tend to overgrow other types of organisms because of the fact that as a group they are more resistant to the acid they produce than are most pathogenic bacteria.

2. Harmful Saprophytic Bacteria.—These organisms decompose milk to form products which are capable of causing gastrointestinal disturbances. It is not necessary that the organism invade the body and cause specific infectious disease. Many of the organisms of this type have marked proteolytic properties. Examples of this group are certain strains of *Escherichia coli*, *B. fecalis alkaligenes*, *Salmonella enteritidis*, *B. lactis*, and certain spore bearers, including the gas bacillus.

Among the harmful saprophytes should probably be included certain cryoflora or organisms which grow only at very low temperatures; and which in consequence develop in frozen milk. The products of the growth of these organisms are believed to be one of the factors in causing the gastrointestinal disturbances sometimes observed when infants are fed milk which has been frozen. The offending substances, however, seem to be destroyed in greater part by subsequent boiling of the milk.

3. Pathogenic Organisms.—Included in the group of organisms capable of producing specific infectious disease *E. typhosa*, *S. paratyphi*, *Shigella dysenteriae*, *M. tuberculosis*, *Corynebacterium diphtheriae*, the *Brucella* group, and hemolytic streptococci.

In raw milk organisms of the lactic-acid group usually grow rapidly and greatly outnumber all other types—indeed, the numbers of the latter may diminish or they may almost completely disappear when a certain degree of acidity is reached. For this reason, milk which tastes sweet may contain more harmful organisms than milk which is definitely sour. When milk is pasteurized most of the lactic-acid producing organisms are killed, whereas certain proteolytic spore bearers survive. When pasteurized milk is poorly refrigerated, the growth of putrefactive organisms is not greatly checked by acid production, so that spoiled pasteurized milk may be more dangerous than sour raw milk.

The bacteria present in milk are not evenly distributed. They tend to become attached to the fat globules so that the cream often has a higher bacterial count than the remainder of the milk. This is especially true of gravity cream. When cream is separated by centrifugalization, the bacteria tend to be thrown down in the sediment.

Methods for Reduction of the Bacterial Count of Milk

Milk intended for infant feeding should be free from pathogenic bacteria and from potentially harmful saprophytes. There are several methods by which the harmful organisms in milk may be reduced in number or eliminated:

1. Prevention of contamination.
2. Heat treatment to destroy such organisms as accidentally may have gained entrance.
3. Acidification.
4. Use of preservatives.

It has been shown that if extraordinary precautions are taken in the production of milk, contamination with harmful bacteria is unlikely to occur. Milk produced under the strictest sanitary conditions, known as *certified milk*, can usually be depended upon to be safe from the bacterio-

logical standpoint. Destruction of harmful or potentially harmful organisms by heat of varying degrees is a safer means of insuring against bacteria. The application of a moderate degree of heat, known as pasteurization, destroys practically all known pathogenic organisms and over 90 per cent of all organisms present in milk. Boiling of milk effects a more nearly complete sterilization than pasteurization, but alters the taste of the milk more. Autoclaving the milk at temperatures above the boiling point, as in the preparation of unsweetened evaporated milk, brings about complete sterilization. The drying of milk has relatively little effect in reducing the number of bacteria below that in the pasteurized milk from which it is prepared.

Acidification of milk, by implanting it with active organisms producing lactic acid or by the addition of a sufficient amount of acid, renders it a poor culture medium for most pathogenic bacteria.

Chemical preservatives such as formaldehyde, benzoic acid, or hydrogen peroxide have been used occasionally to lower the bacterial content of milk. The use of such preservatives is very generally prohibited by law, although it is doubtful whether the small amounts of preservatives necessary for the purpose are really harmful.

Certified Milk

Certified milk is the purest form of raw milk obtainable. It is produced in accordance with regulations prescribed by the American Association of Medical Milk Commissions. The regulations require that the barns in which the cattle are milked shall be of such construction that they may be easily cleaned, that they be screened and that there be adequate ventilation. The cows must all be tuberculin tested at regular intervals and must be free from other diseases as determined by veterinary examination. The milkers must be free from disease and milking must be done with scrupulous care as to cleanliness. After drawing, the milk must be cooled immediately to below 45° F. and kept below

this temperature until delivered. The milk must be bottled at the dairy and must at the time of delivery contain less than 10,000 bacteria to the milliliter.

Certified milk is necessarily expensive, and even with the extreme precautions used in the production of certified milk, contamination is possible. It should, therefore, be boiled before being used for infant feeding. At one time certified milk was the only really clean milk obtainable in cities, and was consequently largely used for infant feeding. With the improvement of the general milk supply, the almost universal introduction of pasteurization, and the availability of sterile preserved milk, the need for certified milk is not so great.

Pasteurized Milk

Pasteurization consists of heating every particle of the milk to at least 143° F. and holding at this temperature for 30 minutes, or heating every particle to 160° F. and maintaining this temperature for 15 seconds. It is then cooled and kept cold until delivery. Failure to keep the milk cold permits the growth of the thermophile organisms not killed by pasteurization.

It is possible to pasteurize milk only when it is reasonably fresh, as milk which is sour is curdled by the pasteurization process. When pasteurization is properly carried out and the milk is protected against subsequent contamination, harmful bacteria are not present. If the pasteurizing process is incomplete, the temperature being too low or the milk not being held at the required temperature for a sufficient length of time, pathogenic bacteria may escape destruction. Furthermore, unless care is taken in the cleanliness of the pipe lines and bottles, contamination subsequent to pasteurization may occur. In general, pasteurized milk is far safer than ordinary grades of raw milk, and properly pasteurized milk is as safe as certified milk. It should, however, be boiled before being fed to young infants. Pasteurization of milk affects the taste

slightly, but not to the same extent as boiling. The heating causes some precipitation of insoluble calcium phosphate, but this loss is of no practical importance. Pasteurization may be expected to cause a loss of approximately 10 per cent of the thiamine and one-third of the ascorbic acid of the milk.

Boiled Milk

Boiling of milk serves much the same purpose as pasteurization, but is somewhat more effective in destroying such organisms as may be present. Boiling also destroys the true bacterial toxins. Boiling of milk for from one to three minutes is sufficient to render it safe from the standpoint of its bacterial content. Boiling affects the taste of milk and also causes the formation of a scum on the surface which consists of coagulated lactalbumin, calcium phosphate, and a few enmeshed fat globules. The scum formed on the milk contains very little of value from the standpoint of nutrition and should be removed before the milk is fed. Boiling, like pasteurization, leads to some precipitation of calcium phosphate and to the destruction of a portion of vitamins B₁ and C, but vitamin C may easily be supplied by the addition of orange juice or tomato juice. Boiled milk is more readily digestible than raw milk because of the change in the physical character of the protein. It is popularly supposed that boiled milk is constipating in its action, probably because diarrhea is less frequent in babies who are fed boiled milk. In view of the distinct advantages of boiled milk, it is advisable that all formulas prepared from sweet cow's milk, whether certified or pasteurized, be boiled before feeding.

Soft-Curd Milks

It is characteristic of cow's milk that, when treated with acid and rennin, its casein coagulates in a fairly firm mass. The resistance of this mass to a cutting edge is referred to as curd tension. The measurement of curd tension has been standardized in that the method of coagulation and

the type of cutting edge have been prescribed. The curd tension is measured in terms of grams required to pull the knife through the curd. The dividing line between hard curd and soft curd is sometimes placed at 30 grams, though a truly soft-curd milk should have a curd tension less than 20 grams. Soft-curd milks are advantageous in that they produce a softer, finer curd in the stomach, and therefore leave the stomach more quickly and are more quickly digested. This advantage, however, is largely for the person past infancy, since all milk for infant feeding should be boiled, and boiling is effective in reducing curd tension sufficiently for the milk to have soft-curd properties.

Natural soft-curd milk is milk that has soft-curd properties naturally, without being processed. Such milk owes its soft-curd property to the lesser amount of total solids, particularly the smaller amount of casein. It has no advantage over ordinary milk diluted to contain a similar amount of total solids.

When milk is homogenized by passage through a valve at pressures of 2500 to 4000 pounds to the square inch at pasteurizing temperature, the fat globules are broken up and their number is increased 200 times or more. This of itself is of little importance in digestion. However, with the increased surface area of the fat, the amount of protein adherent to the fat and unavailable for coagulation is increased. With less casein available for coagulation, a softer curd is formed. Milk homogenized under the conditions stated will have a curd tension less than 20 grams. Homogenization is an increasingly common procedure on the part of dairies.

Soft-curd properties are imparted to commercial milk supplies also by treatment with pancreatic enzymes, the action of these enzymes being stopped at the appropriate point by pasteurization. Another procedure in use in some cities for producing soft-curd properties is the passage of the milk through zeolite, which, under the standard conditions of processing, causes removal of approximately 20 per

cent of the calcium and replacement with sodium. Calcium is necessary for the coagulation of casein and when this amount of calcium is removed, the coagulum is less firm.

Vitamin-D Milk

Milk fortified with vitamin D is available in all large cities of the country and in many smaller ones. Evaporated milk fortified with vitamin D is available at every grocery store. All commercial fortified milks contain a minimum of 400 units to the quart or, in the case of evaporated milk, to the reconstituted quart (415 units to the liter). These are produced by adding to the milk fish-liver oil concentrate, activated ergosterol, or activated 7-dehydro-cholesterol or by feeding irradiated yeast to cows. The vitamin D present in milk is in close association with the fat. Many dairies homogenize their vitamin-D milk, with the result that the vitamin D is evenly distributed through it; thus the amount of vitamin D consumed is always in proportion to the quantity of milk ingested. From homogenized milk, cream cannot be removed for coffee and breakfast cereal. When milk contains 400 units to the quart, the vitamin-D intake of the baby is ample if customary amounts of milk are ingested.

Modification of Milk for Infant Feeding

It has been pointed out elsewhere (Chapters IX and XII) that the essential difference between cow's milk and human milk, as far as the infant's digestion is concerned, is related to the size of the curd in the stomach, and possibly in lesser degree to the buffer value. All successful methods of modifying cow's milk for infant feeding include processing in such a manner that the curd size is reduced. All methods in common use also produce a decrease in buffer value. The most frequently employed procedure is to modify fresh or pasteurized milk by boiling and dilution (Chapter XIV). Evaporated milk has been both heat-treated and homogenized, and is suitable for feeding without further treatment

except dilution (Chapter XV). Fresh milk that has been boiled and suitably acidified is sufficiently altered to be fed without dilution, if desired (Chapter XVI). The dried milks, because of the heat treatment employed in drying, have soft-curd properties, and when reconstituted to the original volume, may be used as would the milks from which they were prepared, but without the necessity for further processing to alter curd properties (Chapter XVII). Other methods for modifying milk for infant feeding are not in common use.

The approximate percentage composition of the various commercial modifications of cow's milk is given in Table V.

TABLE V
PERCENTAGE COMPOSITION OF MODIFICATIONS OF COW'S MILK

FORM OF MILK	WATER	PROTEIN	FAT	CARBO- HYDRATE	ASH	CALORIES TO	
						OUNCE	100 GM.
Fresh, whole	85-88	3.1-3.8	3.4-5.0	4.5-5.2	0.6-0.8	18-24	61-81
average	87	3.5	3.9	4.9	0.7	20	67
Fresh, skimmed	90.5	3.5	0.1	5.0	0.8	10	35
average							
Evaporated, whole	72.7-74	6.4-8.5	7.8-8.2	8.5-10.5	1.4-1.6		
average	73.7	7.0	7.9	9.9	1.5	44	150
Evaporated, skimmed	71	11.2	0.5	14.9	2.4	31	109
average							
Condensed, sweetened	27.5	8.1	9	43.5	1.9	126	330
Powdered, whole	3.5-4	25.8-27.2	26.7-28	36.8-38	5.8-6	141-149	495-510
Powdered, skimmed	1.5-4	32-37.4	1-12	46-52	7-8.4	103-121	325-430
Malted milk	2-3	8-15	7.5-9	70-82	2-4	116-119	387-395

CHAPTER XIV

FEEDING THE NORMAL INFANT WITH WHOLE SWEET MILK MIXTURES

The nutritional requirements of normal infants may be met by the use of simple mixtures of whole sweet cow's milk, sugar, and a diluent, together with food supplements containing vitamin D, ascorbic acid, iron, and thiamine.

Infants vary much in their ability to digest milk. Some young infants are able to take undiluted boiled milk in amounts to satisfy the appetite without suffering from digestive disturbances. Others with lesser digestive capacity require that the milk be diluted. Dilution of the milk serves to make it more readily digestible, and at the same time permits a more generous fluid intake. Dilution is required only for the young infant. Undiluted boiled milk usually can be digested without difficulty at seven or eight months of age if the baby already is accustomed to cow's milk. At four months of age and earlier, the milk need be diluted only one-third to one-fourth. Rarely is it necessary to dilute milk one-half for normal infants, even in the first weeks of life.

Water is the diluent most widely used at the present time. Formerly, when raw milk was commonly prescribed, cereal water, usually prepared from barley flour, was extensively used as a diluent. Starch, because of its colloidal properties, is a deterrent to massive and hard curd formation. Gelatin water also has been used for the same purpose, though in the concentrations commonly employed it seems to have unimportant effects on curd formation. Lime water, in the proportion of 20 per cent of the milk of the formula, also has been used for the prevention of hard curd formation. Sodium citrate, though not a diluent, has been used in the proportion of one grain to the ounce of milk for curd control. The use of boiled milk makes

unnecessary the employment of any of these materials other than water.

In the preparation of whole milk formulas certain general principles should be followed; but for individual infants variations must be allowed dependent on the digestive capacities and appetites. The amount of milk given the young infant should approximate 100 to 130 ml. for each kilogram (1.5 to 2 ounces for each pound), with a general average of 115 ml. for each kilogram (1.75 ounces for each pound) of expected body weight. When the total amount of milk has increased to 1 liter (32 ounces), no further increases are made. The amount of carbohydrate added should bear a relationship to the amount of milk. With customary formulas the amount of added sugar is from 6 to 8 per cent of the total quantity of milk until early in the second half of the first year, when the amount of sugar is gradually decreased as other foods are added.

The amount of water to be added depends on the degree of dilution required to render the mixture readily digestible, and on the total volume of food taken. Essentially the amount of water to be added is represented by the difference between the amount of milk prescribed and the total amount of formula the baby can take easily in twenty-four hours.

The average young baby may be expected to be able to take at a single feeding two to three ounces more than his age in months or an amount in ml. 50 to 60 times the age in months. Although it may be appropriate to prepare the food and offer it to him in this amount, he should not be expected to take exactly this quantity at each feeding. The infant suckling at the breast is permitted a wide latitude in the amount of milk taken, without concern or even awareness on the part of the mother. The bottle-fed baby also should be permitted to vary his intake within limits. During the first seven months the offering of more than 210 ml. (seven ounces) at a feeding or more than a liter (35 ounces) of milk mixture in the twenty-four hours is usually

undesirable. Under no ordinary circumstances at any period in infancy is it desirable to offer more than 250 ml. (eight ounces) of milk at a single meal. As a baby becomes older, he is expected to take many foods other than milk and he has a limited capacity.

The number of feedings in a twenty-four-hour period depends on several factors. Babies are fed on either a three-hour or a four-hour daytime schedule. The best reason for a four-hour schedule is that when feeding time arrives, the stomach is more likely to be empty from the preceding feeding than if the interval were shorter. Young infants usually must have at least six feedings each twenty-four hours in order to obtain adequate food. A six-feeding four-hour schedule necessitates feeding in the middle of the night, whereas a six-feeding three-hour schedule permits an eight-hour interval at night, a desirable objective for most mothers. Although a four-hour schedule often is desirable on the basis of scientific reasoning, it is not so comfortable for the family until the number of feedings in twenty-four hours can be reduced to five. If the four-hour schedule is used from the beginning, the number of feedings should be reduced to five as soon as the capacity of the baby permits. Many babies receiving six feedings of 150 ml. (five ounces) each could take equally well five feedings of 180 ml. (six ounces) each.

The total energy value of the food for twenty-four hours should approximate 110 to 120 calories for each kilogram (50 to 55 calories for each pound) of expected body weight. This goal is easily attained for normal healthy babies. For those markedly malnourished or ill, it may not be possible to give this much food without digestive disturbance. Under such circumstances it may be found that acidified milk formulas are more readily digested.

Construction of the Formula

When starting cow's milk feeding for a baby previously breast fed, it is preferable to prescribe a mixture that has

slightly less energy value than the calculated requirement, with subsequent increases to meet the needs. The rate and extent of increase would be determined by the demonstrated digestive capacity of the infant, evidence of hunger, and rate of weight increase.

A specific example is given of a normal baby, one month of age and weighing 3.6 kg. (8 pounds), who weighed 3.4 kg. (7½ pounds) at birth and who was breast fed for the first month. An allowance of 100 ml. of milk for each kilogram (1½ ounces for each pound) of body weight permits a total of 360 ml. (12 ounces) with an energy value of approximately 240 calories. Sugar added in the proportion of 8 per cent permits the addition of approximately 30 grams (1 ounce), with an energy value of 120 calories. He may be expected to take 90 to 120 ml. (3 to 4 ounces) at a feeding. On a six-feeding schedule and with 100 to 120 ml. (3½ to 4 ounces) at a feeding, the total fluid of the formula would be 625 to 720 ml. (21 to 24 ounces). Consequently the amount of water added would be 270 to 360 ml. (9 to 12 ounces). Thus the formula would be as follows:

Milk 360 ml. (12 ounces)	240 calories
Sugar 30 grams (1 ounce)	120 calories
Water 270 to 360 ml. (9 to 12 ounces)	
<hr/>	
Total 625 to 720 ml. (21 to 24 ounces).	360 calories
100 calories to the kilogram or 45 to the pound.	
Six bottles, 100 to 120 ml. (3½ to 4 ounces) each.	

This formula is adequate in respect to most of the requirements of a formula. It provides sufficient milk, with its protein and mineral salt content, and sufficient carbohydrate. It may be expected to be within the limits of digestive capacity. It should be boiled to make it more readily digestible and to free it from harmful bacteria. This formula is somewhat low in energy value, but it would be a safe one with which to start and the infant might possibly gain weight while receiving it.

If the infant fails to gain in weight and shows signs of hunger, it becomes necessary to increase the amount of food. This is accomplished by adding more milk and, if desired, proportionately more sugar. The amount of milk may be increased to 425 ml. (14 ounces), 115 ml. to the kilogram or $1\frac{3}{4}$ ounces to the pound; the amount of sugar may be increased to 35 grams ($1\frac{1}{4}$ ounces) if the preceding formula has not proved too laxative in effect; otherwise the amount of sugar should remain at 30 grams (1 ounce). The amount of water in the formula would be decreased by the amount of increase in the milk. The formula would now be as follows:

Milk	425 ml. (14 ounces)	280 calories
Sugar	30 to 35 grams (1 to $1\frac{1}{4}$ ounces)	120 to 150 calories
Water	300 ml. (10 ounces)	
Total	720 ml. (24 ounces)	400 to 430 calories
Six bottles 120 ml. (4 ounces) each.		

This formula probably would provide ample energy intake and also would meet all other requirements expected from the formula. If the infant has no digestive disturbance and gains satisfactorily in weight, no change need be made for a few weeks until the food requirements increase because of growth. If, on the other hand, the infant shows reluctance to take as large a volume as this at a feeding, yet shows no evidence of digestive disturbance, the amount of water in the formula should be decreased, the amounts of milk and sugar remaining the same.

By the age of two months the infant might be expected to weigh 4.5 kg. (10 pounds) and might be given the following formula:

Milk	550 ml. (18 ounces)	360 calories
Sugar	35 grams ($1\frac{1}{4}$ ounces)	150 calories
Water	360 ml. (12 ounces)	
Total	900 ml. (30 ounces)	510 calories
Six bottles, 150 ml. (5 ounces) each.		

This formula supplies 113 calories for each kilogram (51 for each pound) of body weight, which may not be sufficient to satisfy the infant. An additional 7 grams ($\frac{1}{4}$ ounce) of sugar increases the calories for each kilogram to 119 (for each pound to 54), an energy intake that should be ample for the majority of infants. Many other changes could be made in adapting the formula to the individual infant. The infant might be one who could take easily as much as 180 ml. (six ounces) at a feeding, and in such case the number of feedings could be reduced to five. If it seemed highly desirable to have a five-feeding schedule and 180 ml. (six ounces) could not be taken, the water addition could be reduced to 210 ml. (7 ounces) and the amount of food in each bottle to 150 ml. (5 ounces); the water requirement would still be met, especially if the orange juice that the infant is to receive also is somewhat diluted.

Still other changes may prove desirable in individual instances. If the baby is constipated while ingesting the formula as prescribed, the type of sugar added could be changed to one with greater laxative properties; within limits the amount of sugar could be increased or the amount of milk could be reduced, or both these measures could be taken and still have the formula meet expected requirements. The reverse of these various measures would apply if the stools were too frequent and loose.

From the preceding discussion it is apparent that no set rules need be, or always can be, followed in the feeding of a normal infant. One must be guided by the appetite, digestion, character of the stools, rate of growth, and general comfort of the baby. Tables giving exact proportions of milk and sugar and the amounts to be taken at different ages are at best only rough guides. The accompanying table is given merely to indicate what the average normal infant might be expected to take at the various ages. In all instances the formula should be made to fit the requirements of the infant; the infant should not be expected to conform to a table.

TABLE VI

OUTLINE FOR THE FEEDING OF NORMAL INFANTS WITH WHOLE SWEET MILK MIXTURES

AGE MONTHS	WEIGHT		MILK		WATER		SUGAR		FEEDINGS		
	POUNDS	KG.	OUNCES	ML.	OUNCES	ML.	OUNCES	GM.	NUMBER	OUNCES	ML.
1	7	3.2	11	330	7	210	1	30	6-7	2½-3	75-90
1	8	3.6	14	420	10	300	1	30	6-7	3½-4	105-120
2	10	4.5	18	540	12	360	1½	35	5-6	5-6	150-180
3	12	5.5	21	630	9	270	1½	45	5-6	5-6	150-180
4	14	6.4	25	750	10	300	1¾	50	5-6	6-7	180-210
6	16-17	7.3-7.7	28	840	7	210	2	60	5	7	210
8	18-20	8.2-9.1	32	960	3	90	1½	45	5	7	210
12	22	10	32	960	—	—	—	—	4	8	240

For additional foods see Chapter XVIII.

Technique of Preparing the Formula

All utensils used in the preparation of the formula should be scrupulously clean and preferably boiled before use. In preparing the formula the total day's feedings are made up at one time. The water is first measured and poured into a clean vessel in which the formula subsequently will be boiled. The sugar is then measured or weighed and is dissolved in the water.

The dry sugars may be weighed on a small balance or scale. More often in the home the sugar is measured in terms of volume, in which case it is necessary to know the volumetric equivalents of a given weight of sugar. Of lactose, dextrose, and the dextrin-maltose mixtures 20 milliliters are approximately equivalent to 10 grams, or two fluid ounces are equivalent to one ounce avoirdupois; 30 ml. of sucrose weighs approximately 30 grams, or one fluid ounce weighs one ounce avoirdupois; 30 ml. of flavored corn syrup contains 30 grams of sugar, or one fluid ounce contains one ounce avoirdupois. The sugar may be measured in a standard tablespoon (15 ml. or one-half fluid ounce) or in a graduated medicine glass; the latter is preferable unless the tablespoons are of standard size. In measuring with a spoon, the spoon should be filled with-

out compressing the sugar, and the content should be scraped level with a knife blade. In measuring in a medicine glass, the sugar should be shaken level by gentle tapping on the glass. When measuring flavored corn syrup by spoon, the syrup should be poured into the spoon and the spoon then should be rinsed in the water; the spoon should not be dipped into the syrup as an extra amount of the syrup will adhere. A convenient method of measuring heavy syrup is to mix it first with an equal volume of water; the resulting thin syrup is easy to manage. In institutions where large numbers of formulas are prepared daily, and where a dry sugar is used, it is often a convenience to prepare the sugar as a thin syrup for use.

After dissolving the proper amount of sugar in the water, the milk is stirred in. The milk should be taken from a fresh bottle which has had the contents mixed by inverting several times. In institutions where the milk may be received in larger containers, it is necessary that the milk be thoroughly mixed before removal for formula preparation.

The mixture of milk, water, and sugar is then boiled for one minute in a saucepan or other suitable container. It is desirable that the mixture be brought to the boiling temperature rapidly and that it be cooled quickly after boiling. Before cooling, the mixture is distributed into feeding bottles, one for each feeding. The bottles are stoppered with nonabsorbent cotton, corks, rubber caps, or cellophane held in place by rubber bands. The bottles are cooled by standing in running water or preferably in water that has been iced; they are then placed in the refrigerator until used.

Periods of boiling longer than one minute sometimes are used, although one minute at the boiling temperature is sufficient to make the mixture bacteriologically safe and to alter the coagulating properties of the casein. Longer periods of heating have certain disadvantages. Milk that

has been heated for twenty minutes or more in a double boiler or in the feeding bottles (after distribution), as sometimes is recommended, has appreciably greater losses of the heat-labile vitamins than milk quickly boiled and cooled. The thiamine loss, particularly, is probably of importance for the young baby.

The Nursing Bottle

Any form of bottle that may be cleaned easily is suitable. Perhaps wide-mouthed bottles are more easily cleaned, but they are somewhat more difficult to handle without spillage than small-mouthed bottles, and the larger nipples are more expensive. Bottles made of Pyrex glass are less likely to break when heated than are those made of ordinary glass. After each feeding the empty bottle should be rinsed and filled with water. Later, when preparing for the feedings for the next twenty-four hours, all bottles are scrubbed with a bottle brush and hot soapsuds, rinsed, and boiled for five minutes.

After use, the nipples should be rinsed and placed in water until time for preparation for subsequent use. They should then be washed thoroughly with warm water and soap, rinsed, boiled, and kept in a clean covered jar that has been boiled. Repeated boiling of rubber nipples shortens their period of usefulness. In a private home where only one baby is being cared for, daily boiling of the nipples possibly may be dispensed with if sufficient care is used in cleaning them and keeping them clean. If boric acid solution is the medium in which they are kept after cleaning, the boric acid should be washed off before placing on the bottle. The life of nipples will be conserved also if, when they are boiled, they are not placed in the water until it has started boiling, and if they are removed from the water promptly after a boiling period of one minute.

Technique of Feeding

Before being offered to the baby the bottle should be warmed to body temperature by immersing in warm water. The temperature of the milk can best be tested by allowing a few drops to fall on the inner side of the wrist.

The hole in the nipple should be of such size as to allow the milk to drop rapidly, but not to flow in a stream. A small hole may be enlarged by burning with a hot needle. If the nipple hole is too small, the milk is obtained with great difficulty and the infant is likely to cease nursing before he has had enough. If the hole is too large, the milk is taken too rapidly, a condition which may lead to choking or to vomiting or both.

The bottle should be held for the baby throughout the feeding. It is best also that the baby be held in a semi-upright position, so as to facilitate the escape of swallowed air. The infant should not be forced to take more food than he wants. On the other hand, he should not be allowed more than twenty minutes at the bottle. Unless the baby is ill or very weak, or the nipple hole is too small, an infant who is hungry will take all the food he needs within ten or fifteen minutes. At the conclusion of the feeding the infant should be held upright, usually against the shoulder, and patted on the back in order to get rid of swallowed air. It may be necessary to hold him for as long as fifteen minutes before the air is brought up.

CHAPTER XV

EVAPORATED MILK MIXTURES

Characteristics of Evaporated Milk

In the preparation of evaporated milk, fresh mixed herd milk is adjusted to a fat content of 3.9 per cent and maintained in an almost complete vacuum at 130° F. until approximately 60 per cent of the water is removed. It is then homogenized in order that the fat may remain distributed through the milk, and is transferred to cans, which are sealed and autoclaved at a temperature of 240° F. for fifteen minutes. The resulting product is sterile and keeps in the sealed cans for long periods without noteworthy physical change. By inverting the position of the can every few months physical changes may be deferred almost indefinitely.

The composition of evaporated milk is constant within narrow limits, regardless of the brand name under which it is prepared and sold. The reason for this constancy is that the product must conform to the definition and standards of the Federal Food and Drug Administration. According to these standards evaporated milk must contain at least 7.9 per cent fat and 25.9 per cent total solids. Actually the fat content is found to vary from 7.9 to 8.2 per cent. It is commonly accepted that dilution of evaporated milk with an equal volume of water results in a product equivalent to the milk from which it was originally prepared. However, the percentage values of the components are on the basis of weight, not volume. The "tall can" has a content of 14.5 ounces avoirdupois (420 grams), or 13 fluid ounces (390 ml.). Starting with evaporated milk having a fat content of 7.9 per cent by weight, dilution with an equal volume of water results in a product with a fat content of 4.16 per cent; the other components are proportionately in excess of the original content. The

dilution factor of 1:1.2 is more nearly correct for approximating the content of the original milk. It is obvious also that the 14.5 ounce can is not equivalent to a quart of milk as sometimes assumed. A fluid ounce of evaporated milk has an energy content of approximately 45 calories; a fluid ounce of a 1:1.2 dilution supplies approximately 20 calories. One hundred milliliters of evaporated milk have an energy content of approximately 150 calories. One hundred milliliters of a 1:1.2 dilution supply approximately 70 calories.

The fat of evaporated milk is not altered in composition, but because of homogenization it is more finely divided than in natural milk. The sugar is practically unchanged by the processing. Because of homogenization and heat treatment the casein is considerably altered, with the result that finer curds are produced in the stomach. As a result of heating, the whey protein also is changed, so that the milk is less allergenic than is pasteurized or boiled milk. Only slight changes occur in the mineral content. A small amount of calcium phosphate is precipitated, but this is easily utilizable after ingestion. Evaporated milk is likely to contain more copper than fresh milk, but the increase is relatively unimportant. The buffer value of evaporated milk is slightly less than that of fresh milk.

As discussed in Chapter VII, evaporation of milk leads to losses of the heat-labile vitamins, ascorbic acid and thiamine. Reconstituted evaporated milk may be expected to contain approximately 6 mg. of ascorbic acid to the liter, which is one-third to one-fourth the amount expected in fresh raw milk and slightly less than one-half that in pasteurized milk. This loss may be ignored because of the universal custom of giving vitamin-C containing foods in addition to the milk. In the case of thiamine, evaporation of the milk produces a loss of approximately 20 per cent, with further losses on storage up to a total of 40 per cent, or possibly more with storage of unusual duration. With our present lack of exact knowledge of the thiamine requirement of infants, it is difficult to state how important

this loss may be. However, it seems clear that the amount of thiamine received by the young infant fed evaporated milk must approach closely the minimum requirement. The feeding of supplemental foods containing thiamine should be begun early.

A large proportion of the evaporated milk on the market in this country has been fortified with vitamin D to the extent of 400 units to the reconstituted quart (416 units to the reconstituted liter). The vitamin-D requirement may be satisfied easily with this amount.

Evaporated milk has an extensive usage in infant feeding. Its uniform composition, sterility and soft curd properties render it well adapted to the feeding of infants. It has the further advantage of being economical. Extensive observations have shown that evaporated milk is nutritionally adequate when supplemented in the usual manner and that its continuous use does not lead to the development of any nutritional disturbance. Evaporated milk should not be confused with *condensed* milk, which is prepared with the addition of approximately 45 per cent of sucrose.

Construction of Formulas With Evaporated Milk

Evaporated milk diluted to its original composition may be used for the preparation of formulas in the same manner as fresh whole milk, the formulas being constructed in the same manner as described in Chapter XIV. Instead of actually making the first dilution and then diluting again for formula preparation, the amount of water to be added is calculated and added at one time. The following table indicates a way in which normal infants may be fed with evaporated milk mixtures. This table is subject to the limitations discussed relative to the use of a similar table for fresh milk mixtures in Chapter XIV.

Because of the changes produced in milk by the process of evaporation, larger quantities and greater concentrations than those suggested in the table may be given to

TABLE VII

OUTLINE FOR FEEDING NORMAL INFANTS WITH EVAPORATED MILK MIXTURES

AGE MONTHS	WEIGHT		EVAPORATED MILK		WATER		SUGAR		FEEDINGS		
	KG.	POUNDS	ML.	OUNCES	ML.	OUNCES	GM.	OUNCES	NUMBER	ML.	OUNCES
$\frac{1}{2}$	3.2	7	150	5	390	13	30	1	6-7	75-90	$2\frac{1}{2}$ -3
1	3.6	8	200	$6\frac{1}{2}$	520	$17\frac{1}{2}$	30	1	6-7	100-120	$3\frac{1}{2}$ -4
$1\frac{1}{2}$	4.5	10	240	8	660	22	35	$1\frac{1}{4}$	5-6	150-180	5-6
2	5.5	12	300	10	600	20	45	$1\frac{3}{4}$	5-6	150-180	5-6
3	6.3	14	360	12	700	23	50	$1\frac{3}{4}$	5-6	210	6-7
4	7.2-7.7	16-17	390	13	660	22	60	2	5	210	7
6	8.2-9	18-20	450	15	600	20	45	$1\frac{1}{2}$	5	210	7
8	8.2-9	18-20	450	15	600	20	45	$1\frac{1}{2}$	5	210	7
12	10	22	450	15	510	17	—	—	4	240	8

For additional foods see Chapter XVIII.

most babies without producing digestive disturbances, particularly to babies three months of age and older. Under three months of age the tolerance of infants for food is more easily overstepped. Such overstepping is made easier by considering equal volumes of evaporated milk and water as being equivalent to whole milk. Such a preparation has a fat content of more than 4 per cent, whereas it is usually preferable to feed babies with milk containing less than 4 per cent of fat.

Evaporated milk diluted with water and without carbohydrate addition has been used successfully in the feeding of both well and sick infants. In order to provide sufficient energy intake more milk must be ingested than with customary types of feeding. The food is higher in protein and fat and lower in carbohydrate than the usual infant's formula. Infants so fed are customarily hungry before feeding, and during the early months they may need seven or eight feedings each twenty-four hours. Successful feeding by this means requires considerable pediatric experience. The relative proportions of milk and water must be adjusted to the individual baby and frequent readjustments are desirable. Infants fed in this manner are reported to grow well and to be active and well muscled. The subcutaneous layer of fat is thin, giving the babies a

lean appearance. Proponents of this type of feeding claim more uniform and satisfactory nutrition and growth than with other types.

Technique of Preparing the Formula

As in the case of fresh whole milk mixtures, the total day's feedings are prepared at one time. The sugar is measured, as previously described, and is dissolved in the requisite amount of boiled water. The required amount of evaporated milk is stirred in. The formula is immediately poured into the clean nursing bottles. No further heat treatment is necessary. The technique of feeding and the feeding intervals are the same as in the case of whole milk mixtures.

CHAPTER XVI

ACID MILK

When cow's milk is soured by the growth of lactic acid producing organisms, or if lactic acid, citric acid, other nontoxic acid, or acid fruit juice is added to the point of curdling, changes are brought about which render the milk more easily digestible. The most important of these changes, and perhaps the only one of significance for the well baby, is the precipitation of casein curds in a finely divided form. A finely divided curd, whether produced by heat treatment of milk, by the addition of rennin, by acidification, or by other means, is more easily permeated by the digestive juices; the milk leaves the stomach more quickly than when larger curds are present, and can be fed in greater concentration than when less efficient methods for modifying the curd are used.

Acidification of milk leads to neutralization of a portion of the buffer substances present, so that when the milk is fed, the degree of acidity attained in the stomach of the infant approximates that attained when human milk is fed. On a theoretical basis this degree of acidity has several advantages. It is sufficient to inhibit bacterial growth, to favor normal functioning of the pyloric sphincter, and to stimulate the flow of bile and of pancreatic and intestinal juices. The contents of the upper intestinal tract are more acid than in the case of sweet-milk feedings—a condition that might be assumed to render certain of the mineral constituents, especially the calcium salts, more soluble and hence to favor absorption. These various factors may possibly be of importance to the sick baby, but for the normal well baby available evidence indicates that the utilization of the various components of the food is equally satisfactory, whether or not the milk has been acidified or

the buffer value changed, provided the curd is in a finely divided form. In the case of the sick baby, the lowered gastric motility and decreased secretion may be compensated in part by acidification of the milk before ingestion.

The acid of acidified milk is quickly absorbed and has little influence on the reaction of the contents of the lower portion of the intestinal tract. The organic acids suitable for acidification of milk are metabolized to carbon dioxide and water, with no effect on the acid-base balance. Mineral acids, on the other hand, such as hydrochloric acid, are not burned in the body, but must be neutralized and excreted. The continued feeding of large amounts of inorganic acid necessarily leads to some disturbance of the acid-base balance, and may deplete the alkali reserves of the body. The small amounts sometimes added to milk may not prove to be harmful.

Because acid milk is not as good a culture medium for most bacteria as sweet milk, formulas prepared from acid milk tend to keep better than sweet milk formulas, even though carelessly prepared and poorly refrigerated. Because of this effect of acid, milk sufficiently acidified has some prophylactic value against diarrhea. However, it is not safe always to depend on this property.

Acid milk is easily digestible, as are all fine-curd milks; in fact, it is so easily digestible that dilution rarely is necessary for feeding even young infants. When formulas are prepared from undiluted milk, the energy values are usually higher than for diluted milk formulas. Little danger of underfeeding exists. Mixtures of this type are of especial value for the feeding of infants with small capacities who vomit when large volumes of food are taken. They are of value also in the treatment of undernourished infants who have large food requirements and reduced digestive capacities.

Acid milk has a few disadvantages. Older infants who are accustomed to sweet milk may refuse sour-tasting milk

at the start; infants who are partially breast-fed do not take acid milk as well as sweet milk, in supplemental or complementary feedings. A few infants secrete gastric juice with unusual acidity; for such infants, acid milk may cause vomiting and at times may have increased laxative effects. The continued use of acid milk beyond the period of infancy is unobjectionable nutritionally; the effect on development of undesirable feeding habits should be considered.

Acid milk, fed in customary concentrations and amounts, gives rise to alkalinity in the lower portion of the intestinal tract, sometimes to a greater degree than when sweet-milk dilution formulas are fed. The increased alkalinity probably is due in part to increased secretion of alkaline intestinal juices, but in a larger measure to the greater amount of milk fed. Because of the greater amount of milk, the stools of infants fed acid milk may at times be firmer and more alkaline. They are composed largely of calcium soaps; the odor is foul rather than sour, and the color may be slightly less yellow than that of stools of babies fed milk more dilute and therefore in smaller quantity.

Lactic Acid Milk

Lactic acid milk, as buttermilk, has been used in infant feeding for at least one hundred and fifty years, and probably longer. It was found empirically that infants with diarrhea often tolerated buttermilk better than other forms of food. Buttermilk is essentially skimmed lactic acid milk, often diluted, and is a by-product of the manufacture of butter. Cream is allowed to sour spontaneously, or the souring is hastened by the addition of a "starter" or culture of lactic acid producing organisms, and the fat is subsequently removed by churning. Buttermilk prepared in this way is likely to be relatively free from harmful bacteria because of the high degree of acidity, but its safety cannot be depended on.

The lactic acid milk used at present for infant feeding is not a by-product of the butter industry, but is prepared especially for human consumption. For the feeding of most infants the fat content of the milk is retained. Either of two methods of preparation may be employed. One consists of souring the milk by means of acid-producing bacteria, the other by direct addition of lactic acid.

Bacterially Soured Milk

For the preparation of bacterially soured milk, any one of several varieties of organisms may be used. Since the chief objective is the formation of sufficient lactic acid, the type of organism selected is immaterial, provided it is nonpathogenic and produces no harmful products. Most of the types of bacteria used die quickly in the intestinal tract. The one most likely to survive, especially if the food contains considerable lactose or dextrin, is the *Lactobacillus acidophilus*. It is possible that the continued growth of this organism in the intestinal tract may exert some beneficial action, but any such effect is difficult to detect clinically.

In most cities dairies prepare bacterially soured whole and skimmed milks that are satisfactory for infant feeding. Bacterially soured milk may be prepared in the home, making use of cultures obtainable on the market. Before attempting to prepare the acid milk, the culture to be used should be tested and a stock culture prepared. This is done by boiling a few ounces of milk, cooling to body temperature and adding to it a small amount of the commercial culture, either liquid or tablet. The inoculated milk is poured into a bottle that has been boiled, the bottle is stoppered loosely and allowed to stand in a warm place for at least twelve hours. The presence of curdling in the milk at the end of this time is an indication that the culture is active.

For home preparation of acid milk, one quart of milk is boiled and cooled to about 80° F. To this is added one

tablespoonful of the milk previously prepared with the culture and the whole is mixed and poured into a clean quart bottle, which is covered with a piece of boiled cheesecloth and allowed to stand in a warm place overnight; or the warm mixture may be poured into a clean vacuum bottle, stoppered, and allowed to stand. The vacuum bottle serves to keep the milk warm. If the milk is incubated at too high a temperature, organisms other than lactic acid producers may grow and impart to the milk undesirable characteristics. Too long a period of incubation results in a product that is excessively acid. The chief difficulty in the home preparation of bacterially soured milk lies in the almost certain eventual contamination of the stock culture with harmful organisms; for this reason new stock cultures should be prepared frequently from pure commercial cultures. Properly prepared cultured acid milk should have a creamy consistency, with no large curds and no separation of whey. The odor should be pleasantly sour, not rancid. Milk containing bubbles of gas is unfit for use. The lactic acid content should be from 0.4 to 0.6 per cent. The curds often are finer than those prepared by the direct addition of acid.

Milk Soured by the Addition of Lactic Acid

Milk acidified by the direct addition of lactic acid does not contain lactic acid organisms, but the presence of these bacteria does not appear to be any great advantage. Acidified milk apparently is as useful as that which has been bacterially soured.

In the preparation of acidified milk, lactic acid (U.S.P. 85 per cent) is added to whole milk in such a proportion as will produce a fine smooth curd, but not in an amount that will cause the curd subsequently to separate. The amount of acid required varies with different milks. For average dairy milk containing not more than 4 per cent fat, the amount of acid required is approximately 6 ml. (1½ fluid drams) to the quart or liter. For most Holstein milk

4 ml. (1 fluid dram) usually is sufficient. One fluid dram of U.S.P. lactic acid is equivalent to approximately 100 drops.

The milk should first be boiled and then cooled on ice or in chilled water. When the milk is cold, the acid is added slowly, drop by drop, with constant stirring. At times it may be more convenient to add the acid to one or two ounces of water, and then to add the mixture to the milk with stirring. At first no effect on the milk is observed, but as the last portions of the acid are added, curdling begins. It is especially important that the last portions of the acid be added slowly, as it is only in this way that fine curds are formed. The final product resembles bacterially soured milk in taste and odor. Unless the milk is thoroughly cooled before the acid is added and unless the acid is added slowly, the curds are likely to be large and clumpy and the preparation will be unsuitable for feeding. If an excess of acid is added, the curds will separate—subsequently if not at once. Larger amounts of acid are not well tolerated by the infant and are likely to cause vomiting. Lactic acid accidentally added considerably in excess of the required quantity causes violent gastrointestinal disturbance in the baby. Amounts of lactic acid less than the stated requirement serve to make the milk more easily digestible, but not to the same extent as when the more appropriate quantity is used.

Several preparations of dried lactic acid milk are on the market. These preparations, when mixed with an appropriate amount of water, are equivalent to freshly prepared products. The dilution factor usually is stated on the label. In general $3\frac{1}{2}$ packed level tablespoonfuls are equivalent to one ounce avoirdupois. A normal dilution of 1 to 8 would be obtained by adding 1 packed level tablespoonful (15 ml.) to 2 ounces (60 ml.) of water.

At times the use of skimmed lactic acid milk is desirable. Acidification of skimmed milk differs in no way from that

of whole milk, except that a smaller quantity of acid usually is required to produce suitable curd formation. Several preparations of dried skimmed lactic acid milk are on the market.

Citric Acid Milk

Citric acid milk is fully as useful and serves the same purpose as lactic acid milk. It is somewhat easier to prepare in that extremely slow addition of the acid to the milk is not essential and the milk need not be quite so cold when the acid is added. Large curds are not easily formed, even though the acid is added rapidly. The curds produced by the addition of citric acid usually are smaller than those formed by the addition of lactic acid. A moderate excess of citric acid is not so toxic as a similar excess of lactic acid. The amount of citric acid required costs approximately only one-fourth as much as does the required amount of lactic acid, when prescription charges are excluded. These various advantages cause citric acid to be preferred by many physicians.

Citric acid is marketed as a dry powder. The amount required to acidify average dairy milk is 3 grams for each liter or quart; for most Holstein milk, 2 grams to the liter is sufficient. Citric acid is used most conveniently as a 25 per cent solution. Each teaspoonful (4 ml.) of such a solution contains 1 gram of citric acid. Solutions of citric acid preferably are kept refrigerated in order to prevent the growth of molds. With appropriate refrigeration, stock solutions keep satisfactorily for several months. If the dry powder is used, it should be put into solution before being added to the milk. One level teaspoonful of the powder weighs approximately 2 grams.

Citric acid milk is prepared in the same manner as has been described for lactic acid milk. The milk is boiled, then cooled. When the milk is cold, the acid is added slowly, with constant stirring. The amount to be added is that which produces definite curdling.

Citric acid milk may be prepared by the addition of the juice of citrus fruits. For this purpose orange juice is commonly employed, and in the amount of 60 milliliters (2 ounces) to the liter or quart of milk. Lemon juice also has been used in the proportion of 25 ml. to the liter or quart.

Other Acid Milks

Acetic acid, as vinegar or as acetic acid U.S.P., has been used by some pediatricians. The proportion recommended is 60 milliliters (2 ounces) of cider vinegar, or 2 teaspoonfuls (8 ml.) of 36 per cent acetic acid U.S.P., to the liter or quart of milk. Acetic acid is readily burned by the body and milk prepared with acetic acid should be as useful as that prepared with citric or lactic acid.

Hydrochloric acid has been recommended as an acidifying agent. In general it seems preferable to use an easily oxidizable organic acid, rather than a mineral acid that must be neutralized and excreted.

A Standard Acid-Milk Formula

Sugar is added to acid milk in the same proportions as to sweet milk, namely, 6 to 8 per cent of the amount of milk for infants up to 6 or 7 months of age and smaller quantities thereafter. With an addition of 6 per cent, the energy value of 100 ml. of the mixture is approximately 90 calories if prepared from average milk, 85 calories if prepared from Holstein milk. The energy value of one ounce is 27 calories from average milk, 25 calories from Holstein milk. Two per cent more sugar adds 8 more calories to the 100 ml. (2.4 to the ounce).

Such a mixture of acid milk and sugar is so easily digestible that it may be fed without dilution to most infants from the time of birth. Moderate dilution is unobjectionable. However, one of the chief advantages of acid milk mixtures is that they may be fed in a concentrated state and that an adequate energy intake is thereby as-

sured. In the case of normal infants, little danger of over-feeding exists, and relatively few conditions arise that make necessary a change in the formula. The same formula is used for months at a time, the only change being increase in the volume, which is determined by the increased demand as the infant becomes older. The formulas are of such a type that individualization is easily possible, though usually unnecessary. The statement is frequently made that no two babies can be fed in the same manner and that the formula must be individualized in each case. This may be true when certain types of formulas are used, but it is not true in the case of foods that meet the requirements in the manner they are met by acidified milk formulas. The success of human milk as an infant food is an excellent example of the lack of necessity for individualization for most babies. After the first weeks of lactation human milk has a relatively constant composition throughout the remainder of the nursing period. The only changes in the feeding of the breast-fed infant are in the total amounts taken and the additions of other foods to the diet.

TABLE VIII
OUTLINE FOR FEEDING NORMAL INFANTS WITH UNDILUTED ACID-MILK MIXTURES

AGE MONTHS	ACID MILK		SUGAR		NUMBER	FEEDING	
	OUNCES	ML.	OUNCES	GM.		OUNCES	ML.
$\frac{1}{4}$	12	360	$\frac{3}{4}$	20	6	2	60
1	18	540	1	30	6	3	90
2	21	630	$1\frac{1}{4}$	35	6	$3\frac{1}{2}$	100
3	24	720	$1\frac{1}{2}$	45	6	4	120
3	25	750	$1\frac{1}{2}$	45	5	5	150
4	$27\frac{1}{2}$	825	$1\frac{1}{2}$	45	5	$5\frac{1}{2}$	165
5	30	900	$1\frac{3}{4}$	50	5	6	180
6-7	35	1050	2	60	5	7	210

For additional foods see Chapter XVIII.

The outline of Table VIII for the feeding of the normal infant, like preceding similar outlines, is only tentative. The proportion of sugar to milk is approximately 6 per cent. The sugar is calculated as a dry powder. If syrup

is used, the allowances for volume must be altered. The total volume taken by the infant will be regulated largely by his desire for food. In starting a young infant on this schedule it may be desirable to use milk from which a portion of the cream has been removed, unless Holstein milk is selected. By the use of an undiluted acid-milk formula babies may have a five-feeding schedule, if desired, at an age earlier than is customary with dilution formulas, because of the greater concentration and energy value of the acid-milk mixture.

The preparation of an acid-milk mixture may be accomplished by stirring into the purchased or prepared acid milk the appropriate quantity of sugar. No further heat treatment is necessary or desirable. If a single formula is to be prepared from sweet milk, a satisfactory procedure is to mix the appropriate amounts of fresh milk and sugar, boil the mixture, cool to a low temperature and acidify with the amount of acid calculated for the amount of milk used. Occasionally batches of flavored corn syrup are encountered that are not neutral, and in these circumstances they may contain enough acid to alter considerably the amount of acid required for proper acidification.

Acidified Evaporated Milk Formulas

Evaporated milk may be acidified in the same manner as fresh milk, except that only about two-thirds as much acid is required for an equal volume of reconstituted evaporated milk, and preliminary boiling of the milk is unnecessary.

Often acidified evaporated milk is fed after dilution with an equal volume of water. When the milk has been acidified and has had added to it an appropriate amount of sugar, it may be fed safely in this concentration to babies three months of age and older. Such concentration in the earlier months, however, is likely to cause gastrointestinal disturbance. For the youngest babies a mixture of one

part of evaporated milk with two parts of water is preferable, even though the mixture has been acidified.

As a convenient variant in the procedure of preparation of a formula, the water, sugar, and acid may be mixed and kept as a stock solution. As needed, it is mixed with evaporated milk in equal volume for infants three months of age and older. The resulting formula is approximately equivalent to whole acid milk with 6 per cent added sugar, and may be used in the same manner. The acid-sugar stock solution should contain 120 grams (12 per cent) of sugar and either 8 ml. of lactic acid or 4 grams of citric acid to the liter of mixture. If citric acid is used, all the ingredients can be mixed and then boiled. If lactic acid is used, the acid is added after the sugar and water mixture has been boiled and cooled. The stock solution keeps well for several days.

CHAPTER XVII

SPECIAL AND PROPRIETARY FOODS

Goat's Milk

Goat's milk has approximately the same composition as cow's milk and may be used in the same manner for infant feeding. The proportion of lactalbumin is slightly higher than in cow's milk. The curd tension of goat's milk is appreciably lower than that of cow's milk. Average values for the percentage composition of goat's milk are stated to be: fat 3.5 to 4.0, lactose 4.8, protein 3.5, and ash 0.7. The vitamin content in milligrams for each 100 milliliters is ascorbic acid 0.65, thiamine 0.047, riboflavin 0.125, niacin 0.30, and pantothenic acid 0.34. The vitamin A content is similar to that of cow's milk.

Most of the claims commonly made for the superiority of goat's milk are unjustified. It is no more readily digested than cow's milk. Goats are less susceptible to tuberculosis than cows, but goat's milk is more likely to be contaminated with the organism of undulant fever. Heat treatment of the milk is desirable for infant feeding, as in the case of cow's milk. Like cow's milk, goat's milk is available in evaporated and dried forms.

In some parts of the world goat's milk is much more commonly used for general human consumption than is cow's milk. In this country cow's milk is available everywhere, and goat's milk is used largely for special indications. The chief indication for its use is in the case of infants who are sensitive to the proteins of cow's milk and develop allergic manifestations when cow's milk is fed. Some of these infants are allergic to goat's milk also, but many are not. No sound basis exists for the substitution of goat's milk for cow's milk in the dietary management of babies who have feeding difficulties.

Macrocytic anemia as a result of the use of goat's milk for infant feeding has been reported, chiefly in the foreign literature. The anemia does not respond to iron therapy, but is relieved by liver or liver extracts. That the anemia is caused by deficiency of one of the components of the vitamin-B complex has been implied. Because the anemia is macrocytic, it appears probable that deficiency of folic acid is involved. With correct dietary supplements, the reported occurrence of this type of anemia need not be a deterrent to the use of goat's milk.

Dried Milk

Both powdered whole milk and powdered skimmed milk are commercially available. A federal definition and standard exists for dried whole milk and all products may be expected to have approximately the same composition. Previous to drying, the milk is adjusted to a fat content of 3.5 per cent. The dried skimmed milks vary in fat content, being prepared from milk with a fat content of from 0.1 to 1.5 per cent.

TABLE IX
COMPOSITION OF DRIED MILK

	WATER	PROTEIN	FAT	CARBO- HYDRATE	ASH	CALORIES	
						OUNCE	100 GM.
Whole milk	3.5	25.8-27.2	26.7-28	36.8-38	5.8-6	141-149	495-510
Skimmed milk	1.5-3.5	32-37.4	1-12	46-52	7-8.4	103-121	325-430

Milk is dried by either a spray or a drum process. In the spray drying of milk, pasteurized milk is sprayed in fine streams into a hot chamber through which a current of hot air is passed; the drying is almost instantaneous and the dried powder falls to the bottom of the chamber. In the hot roller or drum process the milk is run in a thin stream onto steam-heated rollers, either under atmospheric pressure or in a partial vacuum; the dried milk is automatically scraped from the rollers. Milk dried on drums under atmospheric pressure disperses in water less readily than milk dried in the other ways.

Dried milk is not sterile, but it contains few bacteria and none that are pathogenic. The losses of thiamine and ascorbic acid are approximately the same as for the pasteurization of milk; the greater losses that might be expected are avoided by the great rapidity of the drying process. The changes in the casein incident to drying give rise to fine curd formation after ingestion. The fat of dried milk tends to become rancid on storage; for this reason the powdered whole milks and some of the partially defatted milks are packed in an atmosphere of nitrogen. In the large-scale commercial use of dried skimmed milks, this precaution usually is unnecessary and the product is packed in barrels.

Dried whole milk may be reconstituted to its original volume and used for infant feeding in the same manner as fresh milk. In order to obtain the equivalent of the original milk, one part by weight of the powdered milk is mixed with seven parts of water. Four packed level tablespoonfuls of dried whole milk weighs approximately 30 grams (one ounce) avoirdupois.

In order to obtain the equivalent of the original skimmed milk by the use of dried defatted milk, one part by weight of the powder is mixed with nine parts of water. Skimmed milk in its normal dilution is not well suited to the routine feeding of infants, chiefly because of the low energy value. An appropriate formula may be devised, however, by using a larger quantity of the skimmed milk powder than would be used of whole milk powder. A commonly employed formula consists of 20 to 25 grams of dried skimmed milk for each kilogram of body weight (or one-third ounce for each pound), with sufficient added sugar to meet the energy requirement and sufficient water to make the volume of formula customary for the age. A formula prepared in this manner contains less vitamin A than one prepared from whole milk, but otherwise is fully equivalent nutritionally. Some dried skimmed milks, prepared especially for infant feeding, are fortified with vitamin A.

One valuable use of dried skimmed milk is in the preparation of concentrated formulas suitable for the feeding of infants who will take only small volumes and yet who have fairly high energy and protein requirements. In the feeding of prematurely born infants with human milk, better results often are obtained if some dried skimmed milk is added to the human milk before feeding. (See Chapter XXVII.)

Carbohydrate Preparations

The types of carbohydrates used in infant feeding are discussed in Chapter IV. Perhaps twoscore or more of these products are on the market, all intended for use in modifying milk. Even to list them here seems unnecessary. For more detailed information concerning the nature and composition of many of these various products reference may be made to the American Medical Association publication, *Accepted Foods and Their Nutritional Significance*. Despite the large number of names of individual products, the only carbohydrates used in infant feeding are lactose, sucrose, starch, and the starch derivatives, dextrin, maltose and dextrose; also, to a small extent, levulose, present as a part of the invert sugar of such foods as honey and banana. The proprietary names given to many of the preparations, especially the dextrin-maltose mixtures, often tend to give the impression that the foods possess unique properties.

Special cereal foods for feeding infants are discussed in Chapter XVIII.

Condensed Milk

Sweetened condensed milk is prepared by heating whole milk to approximately 90° C., adding sugar, and maintaining the mixture at this temperature for a period of time. It is then condensed in vacuum pans at a temperature of approximately 55° C. to about one-third of the original volume and two-fifths of the original weight. By definition it must contain not less than 8 per cent milk fat and not less than 28 per cent total milk solids. The average percentage composition is shown in Table X.

TABLE X
COMPOSITION OF CONDENSED MILK

	WATER	PROTEIN	FAT	LAC- TOSE	SU- CROSE	ASH	CALORIES	
							FL. OZ.	100 GM.
Condensed milk	27.5	8.1	9	11.5	42	1.9	126	330
1:8 dilution by volume		1.2	1.3	1.8	6.8	0.3	16	42

The product is not heat-processed after canning and is not sterile, the high sugar content being depended on for preservation. It found its place in commerce by virtue of being the form in which milk was first preserved on a large scale. Its chief use is in cooking and candy making. Formerly it had extensive use in infant feeding; its use for this purpose at the present time probably is negligible. In customary dilutions of one to ten and one to eight by volume, sweetened condensed milk is easily digested, so easily that this property may be considered a potential danger. The proportions of the constituents of condensed milk are not those that would be expected to promote optimum nutrition, the milk content being low in relation to the sugar. Because of its easy digestibility, it is often useful in tiding over periods of emergency in which digestion is limited or impaired. Its continued use over long periods is not to be advised.

Malted Milk

Malted milk is prepared by mixing whole milk with liquid from a mash of barley malt and wheat flour, with or without the addition of sodium chloride and sodium or potassium bicarbonate, and maintaining conditions to permit full diastatic activity of the malt. The mixture is then dried. Malted milks vary in their composition. For most of them the content of milk solids is approximately 30 per cent. The fat content varies from 7 to 9 per cent; the protein from 8 to 15 per cent; the carbohydrate from 70 to 80 per cent. One ounce avoirdupois has an energy value of approximately 118 calories. Most of the carbohydrate and some of the protein are derived from the cereal.

Malted milk is a useful food, but it is not of appropriate proportions for infant feeding. The disproportion between milk and carbohydrate is greater than in sweetened condensed milk. At one time malted milk had widespread use in the feeding of infants; at present it has little or none.

Milk-Carbohydrate Mixtures Prepared Exclusively for Infant Feeding

Numerous mixtures of milk and carbohydrate in condensed liquid or dried states, prepared especially for infant feeding, are commercially available. For use it is necessary only to add water. No two of these products are entirely alike and description of them individually seems undesirable. (For their composition reference may be made to the book, *Accepted Foods and Their Nutritional Significance*, American Medical Association.) In general these foods are of two classes: those which simulate human milk in percentage composition, and those which have no such semblance but are more comparable to ordinary cow's milk dilution formulas.

It is apparent that the preparations resembling cow's milk dilution formulas in composition have the same usefulness as cow's milk dilution formulas prepared in the home. It is not so clear that formulas resembling human milk in percentage composition are of as great value as either human milk or ordinary milk formulas. The chief questions involved concern the amounts of nutritional essentials present in relation to the optimum requirements for good growth. The essentials concerned in greatest degree are protein and calcium. In Chapters III and VI attention is drawn to the greater retention by the body of both protein and calcium when the amounts of these fed are greater than those in human milk and are of the same magnitude as exist in ordinary formulas. The argument that the greater retentions are advantageous seems supportable.

In an endeavor to make some of the preparations more like human milk, the milk fat has been replaced by mixtures of fat chosen on the basis that, after being mixed, the combined fat has the same physical constants as those of human milk fat. While at one time a mixture with such physical properties may have had the appearance of being advantageous, newer knowledge of fat utilization indicates no superiority.

To some of the preparations in the group under discussion has been added iron, thiamine, wheat-germ extract, vitamin A, vitamin D, or some combination of these, all in the endeavor to meet known nutritional needs of infants for these materials.

One of the preparations resembling ordinary formulas after dilution, has had the butter fat replaced by corn oil. The purpose in this instance is not to simulate human milk fat, but to provide fat somewhat more readily utilized by delicate and prematurely born babies than is cow's milk fat.

FOODS DESIGNED TO MEET SPECIAL CONDITIONS

Protein Milk (Albumin Milk, "Eiweiss Milch")

Protein milk is a special type of milk mixture, designed by Finkelstein and Meyer for the feeding of infants suffering from diarrhea. The use of protein milk for this purpose is discussed in Chapter XX.

Protein milk is skimmed lactic acid milk to which have been added the curds from whole milk. It possesses the advantages of acid milks in having a low buffer value and small curds. It contains very little of the fermentable lactose and less of the whey salts than whole milk. The fat content also is lower. Protein milk is a poor culture medium for bacteria and is readily digestible. It has a high casein content, which favors the formation of firm stools.

In the preparation of protein milk, one quart of whole milk is warmed to body temperature, rennin is added, and the milk is allowed to stand until coagulated. The curd is cut into small cubes with a knife and the whole is transferred to a cheesecloth bag and allowed to drain for one hour. During this time the curds should not be squeezed as this forces out fat. The drained curd is turned out on a wire strainer and rubbed through with a wooden spoon or potato masher. One pint of lactic acid skimmed milk is mixed with the curds and the whole is again run through the sieve. Water is then added to bring the total volume up to one quart. The average composition of protein milk prepared in this way is: fat, 2.5 per cent; sugar, 1.5 per cent; protein, 3.5 per cent. The energy value is about 13 calories to the ounce.

The preparation of protein milk is time-consuming and it is difficult to prepare a satisfactory product in the home.

Protein milk is prepared commercially on a large scale and is marketed in dried form. Dried protein milk when mixed with a sufficient amount of water gives a product which is as satisfactory as freshly prepared liquid protein milk.

Hypoallergenic Milks

Heat processing of milk alters the protein in a manner to make it less allergenic. Some babies who are sensitive to fresh milk are able to take evaporated milk without reaction. Additional heat treatment reduces the allergenic properties still further. Whole, skimmed, and dried whole milks that have been processed under steam pressure for a long period, are commercially available as "hypoallergic" products. The amino acids lysine and methionine may be destroyed by overheating. Whether such destruction occurs under the long heat treatment necessary to produce hypoallergenic products has not been determined. Until further knowledge is available these products should not be fed indiscriminately or for indefinite periods.

Milk Substitutes

Babies who are allergic to milk products, regardless of how they are processed, often require a food formula that contains no milk. Two types of such foods are on the market. In one of these, soybeans supply the protein base; in the other, a hydrolysate of casein. To the basic material are made appropriate additions of certain nutritional essentials; these additions include minerals, fat and sugar, and in some preparations, vitamins of the B group. For successful use of these preparations, thought must be given to nutritional essentials not included, particularly vitamins. That the soybean preparations, when properly supplemented, support nutrition adequately has been abundantly demonstrated in the case of babies old enough to be receiving fruits, vegetables, and other foods customarily given in the latter part of the first year. For feeding younger babies, and in the absence of all these supplements, the casein hydrolysate preparations are preferable to soybean preparations. Extra additions of B complex, preferably from unpurified natural source, should be given to very young infants in addition to the customary additions of vitamins A, C, and D. The protein of soybean should be fed at a level 50 per cent higher than that of milk protein, in order that all essential amino acids may be present in adequate amounts.

CHAPTER XVIII

THE DIET OF THE NORMAL INFANT

Milk

Milk is the basic food of the infant in that it constitutes the greater part of the bulk of the diet and supplies the larger part of the nutritional essentials which the baby needs. Other foods are added to the diet from time to time, some from the beginning, in order to make the diet nutritionally complete, and also to accustom the baby to varieties in flavors and consistencies of food so that good feeding habits will develop.

The amount of milk required by the infant during the first year is discussed in Chapter XIV. By the end of the first year most babies are receiving a quart or liter daily. During the second year a similar amount is to be considered desirable, though $1\frac{1}{2}$ pints (750 ml.) may meet the needs of many infants. For healthy infants, heat treatment of milk is unnecessary during the second year from the standpoint of increasing the digestibility. However, it is important that the milk be bacteriologically safe, and heat treatment (pasteurization or boiling) is the best way to make certain of the milk's safety.

Cod-Liver Oil

The requirement for vitamin D is amply met by 300 to 400 units of this factor in the form of cod-liver oil or a less concentrated source (see Chapter VII). Since minimum standard cod-liver oil contains approximately 310 units to the teaspoonful (4 ml.), a teaspoonful daily of even a relatively poor grade of oil is adequate. Of more potent cod-liver oils, a proportionate part of a teaspoonful is sufficient. The baby needs vitamin D from the beginning and its administration should be started in the first two weeks

after birth. The dose may be small at the start, but it should be increased rapidly until the full quota is being administered. The dosage of 300 to 400 units daily should be continued throughout the growth period including the summer months unless a special planned program of exposure to sunshine is followed. If more concentrated preparations are used, such as viosterol, a 50 per cent larger number of units should be taken. Babies who receive vitamin D milk do not need additional vitamin D.

Orange Juice and Tomato Juice

The primary purpose for giving orange or tomato juice is to supply vitamin C. For breast-fed babies whose mothers are receiving a good diet, no vitamin C is necessary, though its administration does no harm. Vitamin C is essential for the artificially fed baby and the need exists soon after birth or after breast feeding is stopped. Orange juice, an excellent source of vitamin C, is almost routinely given to infants, but often not as early or in as large a quantity as seems desirable. At least an ounce (30 ml.) of orange juice should be given to the young baby and at least two ounces (60 ml.) by the time the baby has reached the age of three months. If desired, a small amount may be given at first and the quantity may be increased rapidly until the full quota is being taken. From the point of view of digestion, orange juice may be considered merely a 10 per cent solution of dextrose, a material that causes no digestive disturbance in the youngest of babies in amounts much larger than those under discussion. In private practice, apparently many babies are unable to take orange juice without undesirable symptoms, but in institutional practice such an occurrence is rare.

Perhaps the next best substitute for orange juice as a source of vitamin C for babies is tomato juice. In general, natural foods are preferable to pure vitamins or concentrates because of other nutritional essentials they contain, including perhaps essential materials still unknown.

Slightly more than twice as much tomato juice as of orange juice is required to supply the same amount of vitamin C. Ascorbic acid may be given instead of either of these foods; this practice has become common. However, recent studies indicate that the ascorbic acid of orange juice is better utilized by the baby than the same amount given as crystalline ascorbic acid. Potassium citrate given with ascorbic acid increased its utilization, but not to the degree observed when orange juice was given. When ascorbic acid is used, the amount usually given is 25 mg. or a multiple thereof, because this is a standard size of the commercial tablets.

As the baby becomes older, the increased requirement for ascorbic acid may be met usually without an increase of the amount of orange juice beyond two or three ounces daily. The fruits and vegetables that should be added to the diet after the baby is four or five months of age may be expected to contribute significantly to the day's intake.

Egg

When a young baby is receiving human milk, or a cow's milk formula, and supplements of cod-liver oil and orange juice, the diet presumably contains all nutritional essentials in sufficient amount except iron and possibly thiamine (see Chapters VI and VII). Egg yolk is a fair source of both these materials and it has become widely customary to add egg yolk daily to the diet of the infant. Custom differs somewhat as to the age at which this addition is made, but the addition at three months, or between three and four months of age, has a logical basis. A fear that formerly existed that the feeding of egg in early infancy might lead to the development of sensitivity to egg protein seems unfounded in the light of extended clinical experience in instances in which only the yolk has been fed. It is desirable that the egg be cooked. In starting egg-yolk feeding, it is usually preferable to begin with a small amount and to increase the daily amount gradually until an entire yolk is being fed.

For the artificially fed baby, the egg yolk may be added to the formula. The formula is prepared as described in Chapter XIV and is boiled. The egg yolk is separated from the white, and to it are added two or three tablespoonfuls of cold boiled water, and the water and yolk are mixed well. To this mixture is added an equal amount of the hot feeding and the whole is stirred thoroughly. The yolk mixture is then strained into the hot feeding with constant stirring. The entire feeding is then placed over a flame and is brought to the boiling point; it is then ready for bottling.

For either breast-fed or artificially fed babies the egg yolk may be made into a soft custard, which is fed with a spoon. Usually a good time to give it is with the evening feeding. The custard may be prepared by mixing one egg yolk with four ounces of milk (120 ml.) and one teaspoonful of sugar, and heating in a cup by placing the cup in boiling water.

Another procedure is to hard-cook the egg, then to separate the yolk and pass it through a sieve or crumble it with a fork, and make an egg-yolk paste with a small amount of formula. The mixture is then fed with a spoon.

Toward the end of the first year the baby may be given the entire egg. The chief reason it is not commonly given earlier is the possibility that sensitivity to protein of egg white may develop as a result of the feeding. The older baby is not so likely to absorb unchanged protein and therefore he is not so likely to become sensitive. At whatever age the feeding of egg white is started, it should be given cautiously and in small amount at first. When it is evident that no allergic response will occur, a whole egg is given daily. It may be prepared suitably as a soft custard, or it may be coddled, poached, or soft-boiled. An egg may be considered a proper addition to the daily diet throughout the remainder of infancy and for an indefinite period subsequently.

Vegetables

Vegetables supplement the infant's diet moderately in various nutritional essentials, including iron and thiamine, the two materials in which the diet of the young infant is most likely to be deficient. A suitable age for starting vegetable feeding is four months or between four and five months. The vegetables, after being cooked, should be finely divided by passage through a sieve.

Tomatoes, spinach, kale, lettuce, chard, collards, green beans, carrots, beets, peas, asparagus, squash, and celery may be used singly or in combination. All these vegetables are available ready-prepared in cans under various brand names. For the specific nutritional values of the various canned products reference may be made to the book, *Accepted Foods and Their Nutritional Significance*, American Medical Association. The canned vegetables of one brand have been homogenized; whatever advantages such preparations may have, no doubt the chief one is that they may be fed at an earlier age than the coarser foods.

In starting the feeding of sieved vegetables, a small amount is given at first, perhaps a tablespoonful, and the amount is increased until at least two ounces are taken. A good time of day to feed the vegetables is just preceding the afternoon milk feeding. When the baby is between eight and ten months of age vegetables may be given twice daily. Beginning at seven to eight months of age, it is desirable that the baby should become accustomed to foods coarser than those finely sieved. At least one "chopped" or mashed vegetable should be given each day and the amounts gradually increased until none of the foods is finely sieved. Babies who have had only sieved foods up to a year of age are likely to refuse the coarser foods when offered. For the development of good feeding habits it is necessary that the baby become accustomed early to variety in texture and flavor.

Between ten and sixteen months the total daily amount of vegetables served may be three to four ounces, sub-

sequently four to five ounces. Vegetables should have an important place in the diet after the first few months of infancy.

Fruit

Fruits belong in the group of so-called protective foods and contribute important, though moderate, amounts of several nutritional essentials. The content of iron and thiamine is significant, though not large. The addition of fruit to the diet relatively early in infancy, in addition to fruit juice already discussed, is desirable and is a common practice.

Fruits commonly used cooked are prunes, apples, peaches, and apricots. One commercial preparation also contains pears and pineapple. In starting the feeding of fruits the sieved cooked products should be given in small amounts, and the amounts should be gradually increased until approximately two ounces are taken. A suitable starting age is between five and six months. For the same reasons which have been discussed in the case of vegetables, fruit may be fed twice daily beginning between eight and ten months, and at about eight months sieved fruits should be replaced gradually by "chopped" or mashed preparations. The feeding of fruit should be continued throughout the remainder of infancy and subsequently.

Cereal

The feeding of cereal as the baby's first "solid food" has been a custom for many centuries, a custom obviously empirical and having its origin at a time when no scientific knowledge of nutritional requirements existed. Notwithstanding the present trend toward the earlier introduction into the diet of foods other than milk, cereal has maintained its empirical position in relation to other foods, and in practice tends to remain the "first solid food." Many pediatricists are firmly convinced that cereal adds something of importance to the diet and that babies who

receive it thrive better than those who do not. Whatever of importance cereal contains, obviously it is not calories. In the present state of our nutritional knowledge the important contribution of cereals to the diet of the young infant appears to be iron and thiamine. When babies have been receiving only a milk diet, it is reasonable to expect improved nutritional status to result from the feeding of a cereal that contains these two nutritional essentials.

Refined cereals, such as farina, contribute little of nutritional value: the mineral and vitamin contents are negligible. On the other hand, whole-grain cereals make a significant contribution of certain essentials. In Chapter VI in the discussion of iron, and in Chapter VII in the discussion of thiamine, it is pointed out that vegetables and fruits may be expected to supply somewhat greater amounts of both iron and thiamine than the cereals, if the quantities used correspond to those recommended for infant feeding. When the quantities of fruits and vegetables are of the magnitude recommended in the preceding discussion, the young baby usually does not have the physical capacity to ingest the quantities of cereal commonly fed. Because of the greater contribution of vegetables and fruits toward meeting the specific needs of the young baby, it seems preferable to have vegetables and fruits as baby's "first solid foods" and to defer the feeding of cereal until the capacity of the baby has increased to the extent that he can take all three of these foods. Even then, it is less important to feed cereal twice daily, a common practice, than to give vegetables or fruits twice daily.

Some physicians begin the feeding of cereal at three months of age. In conformance with the preceding discussion a more appropriate age would be six or seven months. The cereal should be fed only once a day and the amount given usually should not exceed two fluid ounces of the ready-to-serve material. Whole-grain cereals should be given preference, and in the beginning the preparation should be sieved. Cereal once a day should be considered

an appropriate, though not essential, part of the diet for the remainder of infancy and subsequently.

Several producers of ready-prepared infant foods manufacture cereal foods that have been fortified with iron and the vitamin-B complex. These producers have recognized the specific nutritional needs of the young infant and have provided for their satisfaction in a type of food that has precedence as a "first food." These proprietary cereal foods usually contain significant quantities of calcium, in the form of added calcium salts, or bone meal, or dried milk powder. The added calcium is not needed by the artificially fed baby receiving appropriate amounts of milk, but it may be very useful in supplementing the calcium intake of the breast-fed baby. Since most physicians at the present time prescribe cereal as the infant's first solid food, it is important that the preparation employed be either whole grain or one of the proprietary fortified cereal foods.

Meat

Meat appropriately prepared may be fed at any age. When well cooked and minutely divided, it is easily digested by the young infant. Several of the canned soups sold under various brand names contain finely divided meat. Some of these are known as vegetable soups with cereal and meat; some preparations consist wholly of meat, together with the water of cooking and seasoning. The meats commonly available in such preparations are beef, pork, and lamb muscle meat, beef heart, and liver. The machine processing of muscle meat results in a more finely divided product than can be prepared conveniently in the home. However, it is relatively easy to pass boiled liver through a sieve. Although liver prepared in this fashion in the home is easily digested by the infant, it has not yet become customary to prescribe it for a young baby except for the treatment of anemia. Home-sieved liver and commercially sieved meats may be fed early and, if desired, they may substitute for some of the vegetables; they are a

good source of both iron and thiamine. Although it is common practice to withhold home-prepared meats until the baby is about sixteen months of age, an age when he should have enough teeth to do some chewing, cooked meat, ground, is well tolerated by the ten-month-old infant. Feeding meat occasionally at this age accustoms the baby to food of firmer texture. Ground lean beef, pork, lamb, chicken, or liver may be fed. The meat may be fed two or three times a week and, if desired, it may replace for those days the daily egg of the diet.

Other Foods

Several foods not essential, but properly permitted to the baby, have not been mentioned in the preceding discussions. One of these is bread. Bread, either as toast or zwieback, is commonly given in small amounts at about seven months of age. Perhaps the chief value lies in its contribution toward teaching the baby to chew. Graham or whole wheat crackers also may be used in the same manner. As the baby becomes older, bread is given in somewhat larger quantity, supplying energy and serving as a vehicle for butter.

Certain fruits and fruit juices, not mentioned in the preceding discussion because they are not in common use for feeding infants, may be given not only with impunity but with nutritional advantage. Almost any fruit considered suitable for an older child or adult may be given to the infant when appropriately prepared. Cooked or canned berries, cherries, plums, raisins, and still other fruits may be fed. For the young baby the coarser berry seeds, such as those of the raspberry, and the skins, as of plums for example, should be removed. Cranberries should be fed cautiously or perhaps not at all, because of the benzoic acid content and the effect of this acid in reducing the utilization of calcium. Fully ripe banana, raw or cooked, may be fed at any age; it should be mashed for the young baby. Citrus fruits, including the pulp, may be fed to babies

between one and two years of age. The juices of all the fruits mentioned, except cranberry, may be used. Some of them, such as strawberry, are excellent sources of vitamin C and are useful as variants in the diet and as substitutes for orange juice. Pineapple juice also is a fair source of vitamin C. Sometimes citrus fruits are outside the cost range of low-cost diets and some substitute from among the indigenous fruits or vegetables becomes desirable.

Several vegetables not mentioned in the preceding discussion are appropriate and useful in infant feeding. Potato, mashed or baked, is readily taken and digested by the baby at the same age as other vegetables (see discussion on p. 242). Although potato has not been placed on the preferred list, it is nutritionally superior to refined cereals and is commonly a part of the daily diet of the older infant. Other vegetables that may be given from time to time are eggplant, cauliflower, cabbage, sweet potato, broccoli, turnips, onions, corn, and lima beans. In some parts of the country frijole beans have a prominent place in the diet, and canned frijole beans prepared for infant feeding are on the market. All these foods should have the hulls or coarse fibers, as the case may be, removed by sieving. Rhubarb preferably should not be used for infant feeding because of the oxalic acid content; spinach and beet greens should not have emphasis in the diet for the same reason, though these greens are excellent sources of certain essentials, particularly carotene. Cucumbers, radishes, and green peppers usually are undesirable.

Many variants of the foods discussed, or combinations of these foods, may be used in the baby's diet. For example, cottage cheese may be given to the older baby; rennet custard and ice cream also are good milk foods. Soups may be prepared from vegetables, milk, and butter. Canned soups of vegetables, cereal, and meat are commonly available. Although egg may be served in soufflés or other

preparations in which air is included, it is seldom that an entire egg would be included in a serving; a more compact type of egg preparation is to be preferred.

General Discussion

During the latter part of the first year and throughout the second year the following foods may be considered as basic or more or less essential in the daily diet: at least 1½ pints (750 ml.) of milk; at least one egg or a serving of meat; four to five ounces (120 to 150 ml.) of vegetables; four or five ounces (120 to 150 ml.) of fruit; two to four teaspoonfuls of butter; juice of one orange or four ounces (120 ml.) of tomato juice; one teaspoonful of cod-liver oil, unless vitamin D milk is used. Other foods are added for variety and to satisfy the appetite and energy requirement. These include bread and cereals. Foods not on the basic list ordinarily should not be permitted to replace any of the foods on this list. An example of a daily diet schedule for the second year is as follows:

SAMPLE DAILY DIET FOR THE SECOND YEAR

Breakfast:

- 2 to 3 ounces of fruit
- 2 to 3 ounces of cooked whole grain or fortified cereal with cream and a small amount of sugar
- 1 glass of milk
- ½ slice of toast, or one graham cracker with butter*

Noon:

- 1 egg, or 1 to 2 tablespoonfuls of ground meat
- 1 tablespoonful of mashed or baked potato
- 2 to 3 ounces of vegetable
- ½ slice of toast, or one graham cracker, with butter*
- 1 glass of milk
- A simple dessert, as custard

Evening:

- Milk soup (vegetable purée with milk and butter)
- Custard, or cottage cheese, or ground liver, or egg
- 2 ounces of vegetables
- ½ slice of toast, or one graham cracker, with butter*
- 2 ounces of fruit
- 1 glass of milk (or less if milk soup has been given)

*Fortified margarine is a nutritionally equivalent substitute for butter.

In addition, a glass of milk may be given in mid-afternoon or just before going to bed. The teaspoonful of cod-liver oil may be given in the midmorning, usually about 9 a.m., at the same time that the orange juice is given, or the cod-liver oil may be given at bedtime. In planning a low-cost diet, evaporated milk may be substituted for fresh milk, and tomato juice or the juice of some indigenous fruit that is a good source of vitamin C may replace the orange juice.

When teaching a baby to take a new food or one he dislikes, he should be given at first only a small amount, with gradual increase to a normal serving. Likes and dislikes of food are the result of habit, and habits are acquired relatively easily in infancy. Particularly important in this regard is the mother's attitude toward the food. The mother's likes and dislikes are quickly communicated to the baby from her manner.

Moderate constipation is often readily overcome by increasing the quantity of fruits and vegetables, or in the case of the young baby, by starting the use of these foods earlier than the age suggested.

Small children like to feed themselves and should be encouraged to do so as early as possible. They will need some help with some foods and especially in getting the last of the food from the dish. Young children usually eat slowly and the temptation arises to feed them in order to get the meal finished. Liquid and mushy foods are difficult for the child to eat without spilling and their inclusion in the diet may be expected to prolong the meal time as much as fifteen to twenty minutes. For this reason mothers sometimes object to thin cereal preparations and cream soups. Children 1½ to 2 years of age like crisp or crackly foods, such as corn flakes, crackers, zwieback, and raw celery, lettuce, cabbage, and carrots. At times they may become so entranced with these solid foods that they refuse the liquids. When liquids are refused temporarily, the required amount of milk may be served in thicker or more

solid preparations, such as custards (rennet or egg custard), thickened soups, ice cream, or mixed with other food that is spoon-fed.

Children are highly variable in their desire for individual foods. They may be very fond of a food for a time and then want no more of it for several weeks. Therefore, except for certain basic foods, it becomes desirable not to serve the same food frequently, or, if this must be done, to vary the form in which it is offered.

For such older infants as become hungry between meals or for those who become fatigued easily, extra fruit of some kind may be given advantageously and usually without impairing the appetite for the next meal.

CHAPTER XIX

MALNUTRITION (ATHREPSIA, MARASMUS)

Undernutrition in infants may be of all grades, irrespective of the cause. The infant may be only a little below normal weight for the age and may show no symptoms, or he may have wasted to a skin-covered skeleton, unable to assimilate even proper food and extremely susceptible to infection.

Various names have been used to describe malnutrition in infants such as "atrophy," "hypothrepsia," "athrepsia," "marasmus" or the German term "Dekomposition." By some the term "marasmus" or "Dekomposition" has been applied as indicating a more or less specific condition. It would appear better to consider the various forms of malnutrition as merely stages of the same condition. For convenience of designation, one may consider an infant as essentially normal who is within ten or fifteen per cent of the average weight for his age, birth weight, and length. The term "hypothrepsia" ($\upsilon\pi\acute{o}$ = under, $\theta\rho\epsilon\psi\iota\varsigma$ = nutrition) may be applied to infants twenty to forty per cent below the average weight, and the term "athrepsia" ($a-$ = negative, $\theta\rho\epsilon\psi\iota\varsigma$ = nutrition) or "marasmus" to an infant more than forty per cent underweight. Infants in this latter severe stage of malnutrition present more marked and characteristic symptoms than those who are only moderately undernourished.

The Causes of Malnutrition

The primary and essential cause of malnutrition is the utilization of an amount of food inadequate for the need for maintenance and growth. The inadequacy may be dependent on underfeeding, either as regards total energy value of the food or certain essential components of a com-

plete diet; it may depend on poor absorption or poor internal use of an otherwise adequate diet. Often both these causes are operating in the same child.

UNDERFEEDING

Malnutrition is caused most frequently by inadequate food intake. In the case of the breast-fed baby the supply of milk may be wholly inadequate and the mother unaware of the status of her milk secretion. Less commonly, an infant may be unable to suckle satisfactorily. Weighing the baby before and after several feedings will determine the amount of milk obtained, and subsequent expression of the milk remaining in the breast will determine the baby's efficiency in suckling.

In the case of bottle-fed babies, the formula may be insufficient in quantity or have a low energy value. Sometimes the underfeeding may be more or less intentional on a therapeutic basis, though therapeutic underfeeding for periods long enough to produce malnutrition is never justified. Perhaps therapeutic underfeeding occurs most frequently in instances in which moderate diarrhea is present as a result of chronic upper respiratory infection. The presence of the infection or its relationship to the digestive disturbance may be unsuspected. The digestive disturbance is the outstanding feature, and the dietary management is directed toward decreasing the food to get within the tolerance of the infant. As malnutrition increases, the infant becomes less able to assimilate food and may even have starvation diarrhea. The diarrhea may be interpreted as an indication for further reduction of the food. Instances are encountered in which the therapeutic underfeeding is begun because of vomiting induced by swallowed air, the parents gradually decreasing the food to find an amount that will not cause vomiting.

The erroneous belief that nutritional disturbances in infants are caused by relative or absolute overfeeding with

the different food components undoubtedly has been responsible for much underfeeding. Changing the formula by decreasing one or another food constituent, on the assumption that this or that component has been responsible for causing a specific type of indigestion, is likely to result in underfeeding. When attention is centered on the digestibility of the food and the character of the stools, the energy needs are often overlooked. Occasions arise when it is necessary to decrease temporarily the food intake or the amounts of certain constituents of the diet, but it is to be recognized that food that fails to meet the energy requirement, regardless of how digestible it may be, cannot lead to normal growth and development.

Sometimes mothers have strange ideas as to what constitutes a satisfactory diet for an infant. Cases are encountered in which infants have had a diet of cereal water exclusively for many weeks. Some years ago this general type of feeding was sufficiently common for the resulting nutritional state to be given a specific name in Germany, namely, *Mehlnährschaden*. This name was given on the erroneous assumption that the nutritional injury is caused by starch, whereas actually the cause is starvation.

In an analysis made some twenty years ago of several hundred "difficult feeding cases" coming to the St. Louis Children's Hospital, the primary cause of the difficulty in more than 80 per cent was found to be underfeeding. In many of these the picture was complicated by the presence of infections, but the histories indicated that the majority were underfed for long periods previous to the onset of the infections. During recent years there has been a progressive decline in the number of infants who have been grossly underfed. The better knowledge of nutritional requirements and more general supervision of infants by trained pediatricists, both in the home and in the free clinic, have been responsible for this improvement.

DEFICIENT UTILIZATION OF FOOD

Under the term, decreased utilization, may be included both decreased absorption of food from the intestinal tract and impaired metabolic processes which lead to decreased utilization after absorption. Sometimes both these factors are involved in the same infant. Discussion of the subject may be simplified by considering the primary causes and their relationship to the secondary effects on utilization. The most frequent primary cause is infection; of lesser frequency are congenital anatomical anomalies; still other causes exist, but they are unimportant from the point of view of frequency.

Effects of Infection

Any infection, especially if accompanied by fever, is likely to result in decrease in appetite and digestive capacity. It has been shown experimentally that in the presence of fever, secretion of digestive juices is diminished and absorption from the intestinal tract is impaired. Acute infections result usually in only temporary retardation of weight gain. Chronic infections, on the other hand, may lead to progressive impairment of nutrition. Underfeeding contributes to the occurrence of infections; these infections are more likely to become chronic than in the case of well-nourished infants.

Infections that interfere with nutrition may be either enteral or parenteral. With either localization the gastrointestinal disturbance is likely to include vomiting, diarrhea, or both. The resulting decreased intake and increased loss of food give rise to partial starvation.

With improvement in the care of milk and particularly with the boiling of milk for infant feeding, enteral infections are much less common than formerly. Among the infections affecting the nutrition of infants the chief offenders are those affecting the upper respiratory tract and ears; of lesser importance are infections of the bronchi and urinary tract.

Chronic infection interferes with nutrition even though no fever be present. In fact, chronic low-grade and often unrecognized infection in the ears, mastoid processes, or nasal sinuses is a common cause of gastrointestinal disturbance that leads not only to impaired appetite, occasional vomiting, and some looseness of stools with loss of food, but also too often to therapeutic underfeeding in the attempt to correct the digestive disturbance. These infections with their attendant consequences are a relatively common cause of malnutrition in infants.

Tuberculosis in infants may or may not cause malnutrition. In a high proportion of infants with tuberculosis the presence of the infection would not be suspected from the nutritional state. Only in the more extensive and severe infections is the nutrition appreciably affected. Syphilitic babies also show varying degrees of nutritional disturbance as a result of their disease, depending on the activity and severity of the infection. Many appear entirely normal; others show marked nutritional disturbance. An infant malnourished as a result of syphilis often begins to gain in weight soon after antisiphilitic treatment is started, irrespective of any change in the diet. Further discussion of the influence of infections on nutrition appears in Chapter XXVIII.

Effects of Congenital Anomalies

The failure of infants to thrive is often ascribed to "constitutional weakness." In some instances this term seems justifiable, in others it is used because of inability to state an accurate diagnosis. In certain cases infants fail to do well when the diet is apparently adequate and no evidence of disease can be discovered either during life or at necropsy. Some of these infants are born prematurely and do not appear to have sufficient vitality for independent existence. In such cases the term, constitutional weakness, may be properly applied. In other instances congenital anomalies which account for the nutritional failure are

found at necropsy. The defect may be a rudimentary pancreas, a single small kidney, an atrophic gastrointestinal mucosa, or an anomaly of the vascular or nervous system. These defects interfere with the digestion and absorption of food, or with its metabolism after absorption.

Some congenital anomalies are easily recognized during life, such as malformations of the heart, atresias of the gastrointestinal tract, endocrinopathies and maldevelopments of the central nervous system. These affect nutrition in various ways. In some cases of congenital heart disease the circulation is impaired to the extent that normal metabolism is not to be expected; this is especially true if cyanosis is present. Stunting of the growth of babies with congenital heart disease is a relatively common finding.

In infants with anomalies of the brain, or with intracranial birth injuries leading to spasticity, the maintenance of normal nutrition often is difficult. In many instances the chief difficulty seems to be in getting the infants to ingest the food, but in others the nutrition remains poor when a fairly satisfactory diet is taken.

Endocrine disturbances in infancy are relatively rare. The one most frequently encountered is hypothyroidism, and in this condition, if untreated, growth may be markedly retarded. Uncontrolled diabetes is another endocrine disease in which malnutrition occurs rapidly in infancy because of metabolic failure to make use of the food absorbed.

The occurrence of malnutrition is frequent among babies with cleft lip and palate. In these cases the difficulty lies in the ingestion of food and is the result of the inability of the babies to suckle and swallow, and the ineptitude with which the mother feeds them.

Other Causes

Hygiene no doubt plays a considerable part in the welfare of a baby. However, it is difficult to differentiate the effects of poor feeding and poor hygiene, as the two are

frequently associated. An infant kept in crowded, poorly ventilated, over-heated quarters, or one who is overclothed and receives but little fresh air and sunlight is not likely to thrive in the manner that a baby would under good hygienic conditions.

At the time when the idea of food injury was dominant, the nutritional disturbances of infants were generally attributed to overfeeding with some food component. Overfeeding with cereal was supposed to produce starch malnutrition (Mehlnährschaden); this condition has been discussed under the heading of underfeeding. Overfeeding with milk was thought to lead to malnutrition because of the large amount of protein fed; the nutritional state resulting was known as milk atrophy (Milchnährschaden). This condition was produced, and no doubt still could be, by feeding to young babies relatively large amounts of milk with little or no added sugar. Because of the resulting alkalinity in the lower intestinal tract relatively large amounts of insoluble calcium soaps are formed, giving rise to bulky colon content, which is moved along slowly and from which much of the water is absorbed. The stools are large, grayish-white, and puttylike, and are passed with difficulty. In this country the condition is referred to more frequently as "fat constipation," and the degree of disturbance observed does not generally lead to malnutrition. Such malnutrition as may result would depend on loss of food through excretion, chiefly fats made unavailable by conversion to insoluble soaps. The condition is readily corrected by increasing the sugar intake, or by decreasing the milk, or by both measures combined.

The Symptomatology and Pathology of Undernutrition

The pathology of undernutrition is essentially that of starvation. When the caloric value of the utilizable food is lower than the basal heat output, or when the amounts of specific constituents such as protein and mineral salts in the food are less than the amounts of these components

metabolized in the body and excreted, destruction of body tissue is inevitable. In such instances the fuel need is partly met by the burning of stored fat, carbohydrate, and ultimately protein. Tissues which are destroyed in the natural processes of wear and tear are not rebuilt when the food intake is inadequate.

The utilization of a certain amount of body fat to supply the caloric needs causes little or no damage to the organism as a whole. Fat stored in the subcutaneous tissues and elsewhere is transported to the liver and there utilized as fuel. Stored fat is used only when the amount of available carbohydrate is insufficient. If the amount of fat so utilized is greater than the infant's capacity to oxidize it completely, the ketones formed will not be oxidized. Ketosis and ketonuria result, leading occasionally to definite acidosis.

As the subcutaneous fat is gradually used up, the infant becomes emaciated, the skin becomes shrunken and when a fold of the skin is lifted between the fingers, it is found to be thin and lacking in turgor. Through the atrophied skin may be seen the outlines of the ribs and coils of intestines. The eyes, no longer supported by postorbital fat pads, sink back into their sockets. The infant, however, may not appear sick, although he will, in most instances, be restless and fretful due to hunger.

After the fat depots become exhausted, more vital structures of the body are destroyed to supply energy. The protein of the muscles, of the blood, and of various organs is utilized as fuel. This necessarily leads to general atrophy. The destruction of blood protein results in a decrease in blood volume, and this in turn to poor circulation. For example, it has been shown, in the case of severely athreptic infants, that the volume of the blood passing through a given part of the body in a unit of time may be reduced to as little as one-tenth of the normal amount. Such poor circulation necessarily exerts an effect upon almost every organ in the body. The heart muscle

itself suffers functional disturbance, causing still further impairment of the circulation. The gastrointestinal tract, supplied by atrophied and poorly circulating blood, becomes functionally inefficient, so that digestion and absorption are impaired.

The peripheral vessels, especially those of the skin, tend to collapse and to become constricted. The blood corpuscles pass with difficulty through the narrowed peripheral arterioles and are held back and stagnate on the arterial side. This stagnation of corpuscles, with partial reduction of the hemoglobin, results in a peculiar gray color of the skin.

Infants who have reached an extreme degree of malnutrition have little resistance to infections. They are especially likely to develop rhinopharyngitis, otitis media, bronchitis, and pyelitis. The infection is often of a low grade but very resistant to treatment. Due to poor reaction of the body, local evidences of inflammatory change may be but slight, yet the infection tends to spread and produce a severe degree of toxemia. Infections in the middle ear often spread to the mastoid process without causing much local evidence of the invasion.

Diarrhea from overgrowth of bacteria in the intestine is frequent. Organisms introduced with the food are likely to reach the duodenum, their growth not being inhibited by the very small amount of hydrochloric acid secreted in the gastric juice of the undernourished infant. Furthermore, organisms which ordinarily are found only in the colon are likely to invade the upper intestinal tract and cause vomiting and diarrhea. The mode of invasion is discussed in the subsequent chapter on Diarrhea.

As malnutrition progresses, the red blood cell count diminishes, but the hemoglobin content of the blood is decreased to a greater degree, the anemia being of the hypochromic variety. Red blood cell counts made on blood from skin puncture are often deceiving because of the fact that such blood contains corpuscles held back by a constriction

of the arterioles. A count obtained from the veins at the same time may show a much smaller number of red blood corpuscles.

The leucocyte count is correspondingly high in capillary blood obtained by skin puncture as compared with venous blood. The total leucocyte count is likely to be further elevated as the result of the complicating infections which are so frequently present. The usual white blood count of the athreptic infant is from 14,000 to 16,000 when no infection is present, and rises to 20,000 or 30,000 in the presence of infections. When no infections are present, the lymphocyte percentage is relatively high. In the presence of infections there is a distinct "shift to the left" in the Schilling hemogram.

The protein content of the blood plasma is low, often being no more than 3 or 4 per cent, as compared with a normal of 6 to 7 per cent. As the serum protein falls, there is a tendency for edema to develop, which disappears when the protein is restored.

The heart is small in volume as determined by percussion and x-ray examination. The heart sounds are weak, the pulse is small, easily compressible and tends to be slower than normal. However, it is increased in rate in the presence of fever.

The temperature in the absence of infections tends to be subnormal and may fall to as low as 94 or 95° F. Even in the presence of acute infections, there may be but slight temperature reaction.

The urine shows very little alteration from the normal unless infections of the genitourinary tract are present, when there may be an increased number of leucocytes and bacteria present. Traces of acetone bodies are found during periods of greatly decreased food intake. The total nitrogen and mineral salt output in the urine may exceed the intake of these materials, due to the elimination of products resulting from the destruction of body tissues.

Some infants with severe atrophy show a remarkable tendency to hydrolability, i.e., the water content of the

body is subject to wide and rapid fluctuation. An athreptic infant may gain in weight at an abnormal rate for a short period and become pasty in appearance and edematous, due to water retention. These gains in weight may be mistaken for legitimate gains until the appearance of a definite pitting edema discloses the true nature of the condition. Equally sudden losses in weight occur as a result of loss of water by vomiting or diarrhea. Such losses are accompanied by severe desiccation of the body, with serious symptoms. This phase of the subject is discussed in the chapter on Diarrhea. The athreptic infant often has but little appetite. He may appear hungry and yet take very little food or water when offered.

Vomiting is a frequent symptom. The vomitus may be stained with altered blood in the late stages of severe malnutrition.

Diarrhea occurs with such frequency that it is almost a symptom of athrepsia. It is due in part to the poor functional capacity of the gastrointestinal tract and the inability of the infant to digest and absorb the amount of food required. In even larger measure it is caused by intercurrent infection.

If infections are discovered and are properly treated, and if deficiencies in the diet are remedied, the infant may be expected to gain, provided the malnutrition has not become so extreme that the body has been damaged beyond the possibility of repair. Many infants whose condition seems desperate, ultimately recover to become entirely normal, healthy children. The chief hazard of the undernourished infant is infection. In some infants the flame of life simply seems to flicker out, but in the majority it is an infection that closes the scene.

The Treatment of Malnutrition

Malnutrition is a preventable condition in the majority of cases. Prophylaxis is easier than cure.

Whenever an infant fails consistently to gain in weight at a normal rate, the first step should be to ascertain

whether or not the food intake is adequate in every particular. The caloric value should be checked and a calculation made to determine whether the infant is receiving sufficient protein, carbohydrate, mineral salts, and vitamins. If the diet is adequate and complete and if the infant is taking all of the formula offered but still shows signs of hunger, the volume of the feeding should be increased or the feeding should be made more concentrated by omitting some of the water. Above all, frequent changes in the formula are to be avoided. If the feeding is obviously unsuitable in some respect, the difficulty should be remedied, but there should be a definite reason for the change in every case. There is no surer way of bringing about a condition of malnutrition in an infant than the injudicious changing from one type of feeding to another in the hope of finding something that will exactly agree. Any success which attends such efforts is usually to be attributed to the accidental giving of a sufficient amount of food. When a formula has been selected which meets all the requirements, further changes should not be made except for specific indications.

In the case of breast-fed infants, the total intake of milk should be determined by weighing before and after nursings for a number of days, and if the total amount of milk received is less than that which an infant of the age and size should receive, steps should be taken to alter the nursing regimen so as to insure an increased intake; or else complementary or supplementary feedings should be given.

If no fault can be found in the diet, and if there are no digestive disturbances, a search should be made for possible infections. Any temperature elevation or increase in the leucocyte count should be taken as presumptive evidence of infection. However, it should be noted that some athreptic babies with chronic purulent infections fail to show any significant temperature elevation or localized signs of infection. The leucocyte count in such cases is usually increased.

The examination should certainly include observation of the eardrums and examination of the urine for pus. An intradermal tuberculin test and determination of the Wassermann reaction of the blood give valuable information as to the presence of tuberculosis or syphilis.

Once the condition of malnutrition has developed, the indication is to give a sufficient amount of food to cover the nutritional requirements and yet not more than can be taken care of by the functionally impaired gastrointestinal tract.

The food requirements of undernourished infants are high in proportion to the body weight and approximate those of normal infants of the same age but weighing much more. Thus, an undernourished infant who has weighed 3200 Gm. (seven pounds) at birth, but at the age of four months weighs only 3600 Gm. (eight pounds) requires approximately 120 calories for each kilogram (55 for each pound) of his expected or normal weight, which should be 5700 to 5900 Gm. (12½ or 13 pounds). His requirement would therefore be approximately 700 calories or almost 200 calories for each kilogram (90 for each pound) of actual weight. In some infants the caloric requirement may even exceed this amount in relation to body weight.

Aside from the high caloric requirements, special needs exist for certain specific constituents of the diet in the case of athreptic infants. Relatively more protein is required than in the case of normal infants because of the need for reconstructing destroyed body tissues. It is for this reason that it is often advantageous to add additional protein to the diet of athreptic infants who are being fed human milk. There is sufficient protein in human milk to meet the needs of the normal infant, but not enough for restoring the athreptic infant.

Athreptic infants require a larger amount of mineral salts than normal infants to provide for the reconstruc-

tion of damaged tissues. If the protein intake is made adequate by the use of cow's milk or by the addition of dried milk to human milk, the need for additional salts will be met. When Ringer's or salt solution is given in addition, as usually is done in cases of severe malnutrition, the salts, as well as the water, are useful in helping to meet the needs.

Inasmuch as the capacity of the athreptic infant to digest and absorb food is low, the food necessarily must be one which is easily digested and absorbed. Malnourished infants often are unable to take as large volume of food as normal infants of the same age because gastric motility is impaired so that the stomach empties slowly. For these reasons the food must provide the necessary materials in small volumes, or, in other words, it must be concentrated.

Human milk is easily digestible, but usually it is not possible to give a sufficient amount to meet the energy requirement, and furthermore the quantity of protein in human milk is scarcely sufficient for the needs of the athreptic infant. The energy value and protein content of human milk may be increased by the addition of dried cow's milk. Usually dried skimmed milk is preferable to whole milk for this purpose.

Athreptic infants usually secrete but small amounts of weakly acid gastric juice and often gastric motility is reduced. Such a condition leads to slow emptying of the stomach and offers opportunity for bacterial decomposition of the food in the stomach. Stasis and bacterial activity may be manifested by the vomiting of material having a strong odor of butyric acid. For such babies a type of food is desirable that offers the fewest obstacles to prompt emptying of the stomach. A food meeting this requirement is one relatively low in fat and so prepared that it will have fine curds after ingestion. Acidification of the milk produces a less favorable medium for bacteria and often hastens the emptying of the stomach.

When a baby with marked malnutrition first comes under care, the initial feeding could be acidified boiled skimmed milk containing 6 per cent added sugar. No dilution is required. The amount at a feeding should be small until the reaction of the infant can be noted. The amount can be increased rapidly until a normal volume is being taken or until it is observed that the larger amount is not tolerated. Such a formula is deficient only in energy value, provided the customary cod-liver oil and orange juice and supplements of iron or iron-containing food are given. The energy value is increased as soon as circumstances permit; this may be accomplished by gradual substitution of whole milk for skimmed milk. If the increased fat gives rise to vomiting, particularly if the vomitus is rancid, the fat content of the food should be reduced and the energy value should be increased by the addition of dried skimmed milk, or sugar, or both. If larger amounts of sugar are used, dextrose is the sugar least likely to have a laxative effect.

One should not make the mistake of keeping the food intake too low merely because of the passage of fairly numerous or moderately loose stools. Many undernourished infants gain in weight even though they may not have a strictly normal stool for weeks. The condition of the baby rather than that of the stools is the important consideration.

A sudden increase in the number of stools or excessive vomiting is often due to the onset of an acute infection which may be difficult of detection. The indication in such cases is to search carefully for the infection rather than to alter the character of the feedings. A temporary decrease in food may be necessary, however, during the period in which the infection is being sought and treated. Repeated periods of starvation are especially to be avoided.

Feeding intervals usually should not be shorter than four hours, except in the case of certain prematurely born or

very young infants who will take only small volumes at a feeding. There should be at least six feedings in the day in order to insure an adequate food intake.

Some infants who have become undernourished as the result of an insufficient food intake are ravenously hungry, but a few have become so weak that the appetite fails. The presence of infection also tends to cause a decrease in the appetite. In such instances it may be necessary to resort to gavage or to the administration of food parenterally in the form of dextrose (glucose), either alone or with added amino acids. In some underfed infants the appetite appears to be impaired because of a deficiency of thiamine in the previous diet. In these cases extra amounts of thiamine should be given—as thiamine, or in the form of dried yeast, wheat germ, or vitamin-B complex preparations.

Some athreptic infants take fairly large volumes of concentrated feedings having high caloric values and containing what would appear to be sufficient amounts of all the essential materials and yet fail to gain. There may be no evidences of infection. In such cases the failure to gain is evidently due to incomplete utilization of food, particularly to poor absorption of fat. Thus, we have observed, in the course of a complete metabolic experiment on an athreptic infant, a loss of over 40 per cent of the total caloric value of the food by way of the bowel. This failure of digestion and absorption is in part to be accounted for by the poor circulation dependent on decreased blood volume. Transfusion in such cases is likely to be followed by improved digestion and absorption of food, so that weight gains may occur without further changes in the diet. Transfusion also seems to increase the resistance to infection and often is followed by an improvement in appetite. Keeping the fat content of the diet low is also a useful measure. In some instances either enteral or parenteral administration of amino acids is useful in helping a baby through a critical period.

Indirect transfusions with citrated whole blood are as effective as direct transfusion. A further advantage in the use of citrated blood is that sufficient blood may be drawn from the donor to provide for two or three transfusions at daily or two-day intervals. Repeated small injections are better than a single larger one. The amount of blood given at a single transfusion should be from 20 to 30 ml. for each kilogram of body weight ($\frac{1}{3}$ to $\frac{1}{2}$ ounce for each pound). Amounts larger than this may be injected intraperitoneally, but the intravenous route is usually preferable.

Transfusions should never be given during the stage of acute dehydration or anhydremia. Fluids should always be restored first, as otherwise transfusion may increase the concentration of the blood and result in an exacerbation of all the symptoms of dehydration. For further details as to the technique of transfusion see Chapter XXXII.

Less effective than transfusion in increasing the blood volume and improving the circulation is the intravenous injection of dextrose solution. This results in a temporary increase in blood volume. Hypertonic solutions of dextrose, when injected intravenously, tend to promote absorption from the gastrointestinal tract and also of subcutaneously and intraperitoneally injected fluids. An additional advantage of dextrose is that it provides extra food in readily available form. The strength of solution used may vary from 10 to 20 per cent, the total amounts given being about the same as in the case of blood transfusion. The dextrose given in this way is not completely utilized, as a variable amount is excreted by way of the urine depending upon the strength of the solution and the rapidity with which it is injected.

Dextrose solutions may be administered also by continuous intravenous injection (venoclysis). For this purpose a 5 per cent solution of dextrose, or a mixture of equal parts of 5 per cent dextrose and Hartmann's physiological

buffer salt solution (see p. 301), or Ringer's or physiological salt solution, may be used. The 5 per cent dextrose solution or the combined dextrose and saline solution may be given at the rate of 8 to 16 ml. an hour for each kilogram. Too rapid injection may lead to chills, temperature rise, and edema. The injection may be continued day and night for several days. By means of such an injection sufficient calories are supplied to tide the infant over a period when he may be receiving little or no food by mouth. The addition of Hartmann's or of Ringer's solution supplies necessary mineral salts to replenish the depleted mineral reserves of the body. Such injections are especially valuable in those cases in which there has been severe diarrhea associated with desiccation of the body and concentration of the blood (anhydremia). For further details see table on p. 484.

The athreptic infant, as already mentioned, shows a tendency to hydrolability and may, at any time, become dehydrated as the result of vomiting and diarrhea and failure of the body tissues to retain water. Unless sufficient fluid is supplied, the symptoms of anhydremia are likely to be superimposed on those of athrepsia. In order to prevent anhydremia, water should be administered to athreptic infants in sufficient amounts to prevent any desiccation. It is not always possible to give sufficient water by mouth, so that resort must be had to subcutaneous or intraperitoneal injection. Furthermore, water may not be retained unless the additional mineral salts of saline solutions are given at the same time.

In most cases of athrepsia, infections occur. In the case of an undernourished infant suffering from a chronic infection, it is often impossible to determine whether the nutritional state is the result of infection, or vice versa. The two go hand in hand, the one predisposing to the other. So long as infection exists, it is difficult to restore the infant to a state of satisfactory nutrition, even with feedings

which otherwise would be adequate. Furthermore, until the nutrition is improved, infections are not likely to clear up except as they may be influenced by chemotherapy. This explains, in part, the high mortality among infants suffering from extreme undernutrition.

The athreptic infant should be examined regularly for evidences of infection. Some athreptic infants develop a low-grade otitis media which is difficult to detect on otologic examination. In others, middle ear infections spread to the mastoid antrum with resulting exacerbation of constitutional symptoms, especially vomiting and diarrhea, although local manifestations in the mastoid may be but slight. The additional infection may throw upon the already weakened infant a strain beyond his powers to resist. For a fuller discussion of the relationship of infections to nutrition and the treatment of infections, see Chapter XXVIII.

CHAPTER XX

DIARRHEA

General Considerations

In past years, the diarrheal diseases have accounted for almost as many deaths among infants as all other causes combined. Mortality from this cause has shown a progressive decrease, but even at the present time diarrhea has a prominent place in published mortality figures for infants who have survived the neonatal period. The actual number of deaths attributed to diarrhea is probably not so great as vital statistics would indicate because of the fact that infants seriously ill from any cause are likely to develop diarrhea as a terminal symptom and when the original disease has been undiagnosed, diarrhea is given as the cause of death.

Due to a number of factors infants are much more likely to develop diarrhea than are older persons. The gastrointestinal secretions of the infant differ in composition from those of older persons. The gastric juice contains less pepsin and rennin and very much less hydrochloric acid. The gastric juice of the infant is well adapted to initiate the processes of digestion when human milk is fed. Small curds are formed in the stomach and the contents become sufficiently acid to inhibit bacterial growth. Probably as the result of this acidity, the stomach and upper portion of the intestinal tract are normally free from bacteria capable of causing gastrointestinal disturbance. In consequence the baby exclusively breast fed rarely suffers from severe diarrhea. There are, however, circumstances in which the gastric and intestinal secretions may be diminished.

Any infection accompanied by fever is likely to result in a decreased secretion of gastric acid and of the pancreatic and gastric enzymes. A similar result is brought about by

exposure to high temperatures. Some types of parenteral infection are more likely to lead to diarrhea than others, even though the elevation of temperature may be the same. For example, infants suffering from rhinopharyngeal infections and otitis media are in general more prone to develop diarrhea than those suffering from pyelitis.

Infections outside the gastrointestinal tract are the most frequent underlying causes of diarrhea occurring in infants who are breast-fed or who are receiving well-constructed cow's milk formulas; such infections are most common during the winter months. Enteral infections are a frequent cause of diarrhea in artificially fed infants living under poor hygienic conditions, especially during the summer months.

An infant who from any cause has become badly undernourished, is likely to have lessened gastric secretion. In such cases, the contents of the stomach may not be sufficiently acid (even when human milk is fed) to bring about bacterial inhibition, and as a result organisms such as those of the colon group, which are not ordinarily inhabitants of the duodenum and stomach, may be present. When the breast-fed baby receives, in addition, articles of food which are bacterially contaminated, some potentially harmful microorganisms may survive in the intestinal tract.

The infant is incapable of digesting many foods which may be digested easily by the adult with stronger gastrointestinal secretions, and in consequence the diet of the artificially fed infant must be limited largely to milk with added sugar during the earliest months and to relatively simple foods throughout infancy. Foods less easily digestible may irritate the intestinal tract mechanically, or in some instances may serve as a means of introduction of harmful bacteria. Both milk and sugar solutions are excellent bacterial culture media, and milk, unless obtained under clean conditions and heat-treated before use, contains numerous bacteria, some of which may be of a harmful type. Even with a carefully selected and well prepared

diet, the artificially fed baby is in general more susceptible to diarrhea than the breast-fed baby. The reason is not wholly clear. One reason appears to be dependent on the high buffer capacity of cow's milk as compared with human milk. The small amount of gastric acid is neutralized to a greater extent and less acid is available for bactericidal effect. With infection or illness the amounts of acid and digestive ferments are still further decreased and the small margin of safety disappears more quickly than with breast feeding. Perhaps in the private practice of pediatrics diarrhea is little (if at all) more frequent among artificially fed babies than among those who are breast-fed, but the proportion of babies cared for in private practice is small. In this group environmental factors are likely to be more favorable for the general welfare of the infant. These factors are particularly important for the artificially fed infant as they affect the preparation, handling, and administration of the food, whereas in the case of the breast-fed infant the milk supply is not subject to these environmental hazards. In the case of babies with undernutrition, gastrointestinal secretions are likely to be diminished as a result of the nutritional state and diarrhea is more likely to occur as a consequence of illness or overfeeding. Undernourished babies tend to have increased susceptibility to infection.

Considerable evidence exists that many of the diarrheas of infancy are the result of the growth *in the upper intestinal tract* of organisms which are normally present only in the lower bowel. The chief exponents of this viewpoint have been Moro, Plantenga, and Arnold. Strong confirmatory evidence has been obtained by a large number of other investigators. According to this viewpoint we may explain the effects of overfeeding or the feeding of indigestible material as due to the accumulation of unabsorbed bacterial culture media in the intestinal tract rather than to any specific harm caused by the foods themselves. It must be admitted, however, that certain types

of foods may have a laxative effect. A concentrated solution of some of the sugars may act as a hydragogue laxative, in addition to any effect produced by bacterial action on the excess of sugar. Certain of the lower fatty acids, such as butyric acid, produced by bacterial action in the stomach during gastric stasis, are a cause of vomiting and, if passed into the intestinal tract, are irritants and therefore laxative. Foods which act as irritants in the intestinal tract increase the secretion of mucus, and with decreased absorption a hydragogue effect results.

Bacteriological examinations of the stomach and duodenal contents of infants suffering from severe diarrhea have revealed the fact that organisms of the colon group are usually present in large numbers. These organisms are not found in the case of normal infants, although certain other organisms, especially harmless cocci, may be present. The significance of this rather uniform finding is not entirely clear. It is, of course, possible that the colon bacilli reach the upper portions of the intestinal tract *because* of the fact that a disturbance is present. It seems, however, more likely that their presence is an important factor in bringing about the gastrointestinal disturbance.

Colon bacilli which may exert no harmful effect when growing in the lower bowel may lead to serious disturbances if present in the upper portion of the intestinal tract. The effects may be produced in two ways: by actual invasion of the body or by the elaboration in the intestine of toxic products which, when absorbed, are capable of producing serious symptoms. It has been shown that colon bacilli present in the duodenum may pass through the intestinal mucosa and appear in the thoracic duct lymph in the case of animals in which the duodenal contents have been kept alkaline. The colon bacillus is not infrequently found in blood cultures taken from young infants or those with severe gastrointestinal disturbances associated with the presence of colon bacilli in the stomach and duodenum.

Plantenga has shown that the administration by mouth of the filtrates from broth cultures of *E. coli* is followed by vomiting and diarrhea, but that the same symptoms are not produced when the filtrates are introduced rectally. The exact nature of the products responsible for the production of the symptoms is not known. Peptones and the ptomaines, cadaverine and putrescine, do not produce the symptoms. The symptoms do not appear to be due to endotoxins, for when killed cultures of *E. coli* are injected, the symptoms do not appear. It has been suggested that histamine and other amines may be the toxic agents. Some strains of *E. coli* are capable of producing large amounts of histamine, or a histamine-like substance; other strains produce none. Histamine, when injected even in minute amounts, causes vomiting, diarrhea, hydrolability, and a condition resembling shock, but when introduced into the intestine does not usually cause toxic manifestations. This lack of toxic symptoms is explained by the fact that detoxication occurs on passage through the liver and possibly to some extent on passage through the intestinal mucosa, but whether such detoxification occurs completely when the intestinal tract is damaged, or when large amounts of histamine are produced, is a question.

It has been believed very generally that acids produced by the bacterial decomposition of carbohydrates are responsible for the production of diarrhea. The fact that diarrheal stools are often fermenting and acid has been the basis of this belief. In any condition in which unabsorbed fermentable carbohydrate reaches the lower bowel, fermentation by the abundant flora invariably occurs; therefore, the fact that the stools are acid is in itself not necessarily indicative of the presence of much acid in the upper bowel. Some acids are irritating, but it is doubtful if acid production in itself is often the cause of diarrhea. A reasonable amount of acid in the upper intestine is advantageous rather than otherwise, and only a great excess appears to be harmful. Actual observations on infants

have usually shown a lesser rather than a greater degree of acidity in the upper intestinal tract in cases of diarrhea. Furthermore, it has been demonstrated experimentally by Lloyd Arnold that alkalization of the upper intestinal tract favors bacterial growth. When this condition is reproduced in animals by feeding an alkaline buffered solution, the introduction of colon bacilli and other organisms results in severe diarrhea. Acid in the lower bowel may have more effect in delaying absorption and aggravating diarrhea than it does in the upper intestine. Diarrheal stools frequently are sufficiently acid to excoriate the skin of the buttocks; acid of such concentration must produce irritation of the colon also.

Although it appears likely that invasion of the upper intestinal tract by bacterial flora of types found ordinarily only in the lower bowel is a common cause of the serious forms of infantile diarrhea, it should not be inferred that such is the only cause. Other types of bacteria may cause diarrhea when introduced by way of the mouth and which in this way gain a foothold in the intestinal tract, as for example *E. typhosa* and members of the *Salmonella* group. Typhoid produces relatively mild symptoms in the infant as compared with those of older persons. Diarrhea is the chief and often the only symptom. The *Salmonella* may cause food poisoning with diarrhea by means of toxic products produced through growth in food or they may cause definite enteritis because of their pathogenic properties. Some strains of streptococci also lead to enteritis. Amebiasis seems to be very uncommon in this country among children under two years of age. More important than any of these is the dysentery group of organisms, including the "true" dysentery bacillus of Shiga and the paradysentery group. These organisms are the causative factor in bacillary dysentery which is an acute infectious disease characterized by superficial necrosis or ulceration in the colon and lower ileum. Diarrhea is one of the

symptoms of dysentery. Bacillary dysentery is discussed in detail in Chapter XXI.

Staphylococcus aureus is a pathogenic organism that can produce diarrhea, not by its implantation in the gastrointestinal tract, but by producing an exotoxin when growing in food. This organism is the most frequent cause of food poisoning among adults, but it is not often a cause in infants. Instances have been encountered in which the milk supply had *Staphylococcus aureus* as its dominant organism. Boiling such milk for the usual period does not affect the toxin.

Besides the definitely pathogenic organisms certain saprophytic bacteria may decompose food with the production of irritating substances. When food so decomposed is eaten, or if the bacteria in question are introduced into the intestinal tract, where they may decompose food remnants present, diarrhea may be the result. Undoubtedly a number of organisms may fall in this classification, many of which are not clearly defined. Some are definitely proteolytic and it is believed that the products of protein decomposition are responsible for the symptoms. Others appear to produce irritant substances from carbohydrates. Among saprophytes suspected of being the cause of diarrhea are the members of the *Proteus* group, several types of organisms described by Flügge, possibly the gas bacillus, and a large group of nondescript organisms which may be found in partially spoiled food. Diarrheas produced by organisms of this type are usually of short duration and are not frequently accompanied by severe manifestations.

Certain virus infections cause diarrhea. Prominent in this category is epidemic diarrhea of the newborn. The infection presumably is enteral, although the intestinal mucosa shows only congestion. Epidemic diarrhea of the newborn is highly communicable and is difficult to eradicate from nurseries.

Factors other than the food and bacterial conditions in the gastrointestinal tract may lead to diarrhea. In some

infants hypertonicity and hypermotility of the entire gastrointestinal tract are constitutional characteristics. This special condition of "gastroenterospasm" is discussed in Chapter XXV. Infections at times appear to disturb the balance of the autonomic nervous system, with resulting increased peristalsis irrespective of the character of the food.

Food allergy may be a factor in the production of diarrhea. The offending substance is more likely to be the protein of cow's milk, or of eggs, but may be the protein of cereals, vegetables, or other additions to the diet. In rare instances mother's milk leads to gastrointestinal symptoms due to the passage of foreign proteins into the milk when the mother has taken these in her food.

That underfeeding may lead to diarrhea would appear, at first sight, unlikely, yet this is a fairly frequent cause. Starvation or hunger diarrhea is likely to occur in infants who have been quantitatively or qualitatively underfed for considerable periods of time until they have become undernourished, rather than in healthy infants subject to short periods of starvation or decreased food intake. Too low a total caloric intake or a deficiency in protein, carbohydrate, mineral salts, and certain of the vitamins may bring about the condition. In the presence of hunger, hypermotility of the gastrointestinal tract is observed and experimental evidence seems to indicate that such hypermotility is a direct result of lowering of the blood sugar content. Furthermore, starvation or underfeeding, if prolonged, results in a decrease in the gastrointestinal secretions and also in a decrease in the blood volume and in the volume flow, so that absorption from the intestinal tract is impaired. Hunger diarrhea differs from the other types in that the stools, though numerous, contain relatively little food material. Gastrointestinal colic or cramps are frequent accompaniments.

An inadequate water intake should be included in the category of underfeeding. When the water intake is in-

sufficient to cover the water output by way of the urine, stools, respiration, and perspiration, the result is desiccation of the body, or anhydremia. One of the results of anhydremia is diarrhea, which is often accompanied by symptoms of intoxication.

Diarrhea should not be considered as a disease *sui generis*, but as a symptom resulting from a variety of causes. Many infants with diarrhea also suffer from other manifestations as, for example, vomiting. Indeed, vomiting and diarrhea are often part and parcel of the same condition.

Symptoms of Diarrhea

Diarrhea occurring in well-nourished infants as the result of temporary overfeeding, contamination of the food with nonpathogenic saprophytes, high external temperatures, or the presence of parenteral infection is usually mild in character, of short duration, and unaccompanied by severe constitutional symptoms. The stools are increased in number, and soft in consistency. The color usually varies from yellowish green to bright green. Soft, white soap curds are present if any fat is contained in the diet. Diarrheal stools are likely to be acid in reaction if sugar is being fed. Some glairy mucus is usually present and is an indication of intestinal irritation. Blood and pus are not present in cases of mild diarrhea.

Vomiting frequently accompanies diarrhea. It is seen especially in those cases in which the diarrhea is due to parenteral infection, in which instances vomiting often precedes the first evidences of diarrhea. Temperature elevation is slight in the uncomplicated diarrhea of older infants. Any considerable rise of temperature should arouse the suspicion of the presence of a parenteral infection, or of bacillary dysentery. Some loss of weight is inevitable in all cases of diarrhea, but in the milder types little weight loss occurs. A marked weight loss is indicative of a more severe or toxic type of diarrhea. Usually some loss of

appetite is observed. Infants with diarrhea are fretful and irritable, and appear to suffer from abdominal pain, but severe toxic manifestations are absent in the milder forms.

The severer forms of diarrhea are seen especially in undernourished infants, young infants, and those suffering from certain types of acute parenteral infections. To the severe forms of diarrhea, the term "alimentary intoxication" is sometimes applied, because of the toxic manifestations accompanying the gastrointestinal disturbance. The stools are numerous and may be as many as 15 or 20 in the course of twenty-four hours. At the onset they do not differ a great deal in character from those seen in the milder illnesses. They are green, acid in reaction, and contain food remnants and mucus. Later they may consist of little else than brown, watery fluid, often alkaline in reaction. The total volume of the stools may be very great and seemingly out of all proportion to the food and fluid intake. As the stools may be composed largely of fluid, which is absorbed by the diaper, the total volume may not be appreciated. When the stools are collected in a basin and measured, it may be found that the daily volume actually exceeds the fluid intake. Because of the character of the stools, severe diarrhea is sometimes designated by the term "cholera infantum." Small amounts of blood are occasionally present in the stools, but any considerable amount of blood should arouse the suspicion of bacillary dysentery infection. Gross pus is not seen, but leukocytes may be fairly numerous on microscopic examination. Loss in weight is sudden and severe and is accompanied by the development of the symptoms of anhydremia.

The symptoms of epidemic diarrhea of the newborn do not differ essentially from those described for diarrhea from other causes. The onset is sudden. If any fever is present, it is only moderate. Anorexia is present. The stools are watery and are discharged explosively. Because

of the severity and persistence of the diarrhea, the mortality is high, sometimes as high as 50 per cent.

The symptoms of severe diarrhea are to be explained as the result of the combined action of a number of factors, chief among which are anhydremia, acidosis, and bacterial toxemia.

Bacillary dysentery differs in a number of important respects from the other types of diarrhea and is therefore discussed separately. (See Chapter XXI.) It should, however, be pointed out that at the onset the symptoms of bacillary dysentery may be entirely indistinguishable from those of other forms of diarrhea.

The Effects of Diarrhea on the Body

Diarrhea, if severe or prolonged, may result in serious damage to the body or in death. The chief effects are:

1. Diminished absorption of food resulting in partial starvation.
2. Loss of water (anhydremia).
3. Loss of mineral salts, especially fixed bases (acidosis).
4. Toxemia from intestinal bacteria.

Diminished Absorption of Food

In the presence of diarrhea, the food intake is usually decreased as a therapeutic measure, and such food as is taken is only partially digested and absorbed; hence the actual amount of food reaching the tissues may be insufficient to meet the fuel needs of the body. In such circumstances, the body tissues themselves are necessarily consumed for fuel. The using up of some stored body fat and carbohydrate leads only to a moderate loss of weight and no serious consequences. If, however, partial starvation is long continued, serious malnutrition or athrepsia is certain to result. In the presence of diarrhea, fats are especially likely to escape absorption, as much as 25 or 50 per cent being lost by way of the bowel in the form of fatty acids, neutral

fats, and soaps. A considerable portion of ingested carbohydrate may be destroyed as the result of bacterial action, with the production of acids, carbon dioxide, and other substances. The loss in this way may amount to over 50 per cent of the intake. Proteins are usually fairly well absorbed, even in the presence of diarrhea, a loss of more than 15 per cent of ingested protein being unusual. The fat-soluble vitamins, like fats, are likely to escape absorption.

Anhydremia

In the presence of diarrhea, water absorption is poor, which accounts for the looseness of the stools. The amount of water lost in this way may be very large and is at times as great or greater than the total fluid intake. As a result of this water loss, desiccation of the body tissues and concentration of the blood occur. These processes lead to a serious train of symptoms. The condition of general desiccation of the body has been referred to as "dehydration," "exsiccosis," or "anhydremia," the latter term being descriptive of conditions present in the blood.

Anhydremia may occur in the absence of diarrhea, due to a diminished intake of fluid, or as the result of vomiting from any cause. Anhydremia is of more frequent occurrence and is of a more severe degree during warm weather because of the fact that there is then a greatly increased loss of water by way of the skin and lungs.

The symptoms of anhydremia are largely dependent on a decrease in the volume of the blood and are, therefore, especially likely to occur in the case of athreptic infants in whom the volume of blood is already diminished. Some of the symptoms of the two conditions are identical.

One of the first indications of anhydremia is a loss of body weight, which often is extreme and rapid. A small infant may lose half a pound or a pound in a single day, the loss being chiefly water. Coincident with the loss in

weight, the appearance of the patient changes greatly. The features become sharpened, the eyes sunken and often fixed in a far-away stare. Later the conjunctivae lose their luster and are coated with a lusterless film. The eyes are likely to be turned up under the half-closed upper lids. The fontanel is depressed; the skin has a peculiar pallor and often a characteristic grayish color like that of wet ashes. This color of the skin is dependent on arteriolar constriction with piling up of red corpuscles in the capillaries. The capillary blood count is distinctly higher than that of the venous blood. The arteriolar constriction is one of the results of a diminished blood volume. The skin over the body is dry and may be picked up into folds which remain an appreciable period before flattening out. The lips are dry, parched, and often of a peculiar cherry-red color. The mouth is held partly open; the tongue is dry. (Fig. 8.)



Fig. 8.—Anhydremia and acidosis. (“Alimentary intoxication.”)

The pulse is small, sometimes almost imperceptible, often rapid and irregular. The volume flow of the blood is greatly diminished as the result of its concentration. The blood is thick and does not flow easily. When the blood is centrifuged, relatively little serum separates. The concentration of protein in the serum is invariably high and the water content low. Leukocytosis of a moderate degree is frequently present.

The urine is very scanty, and highly concentrated, and contains numerous granular casts and some albumin. Occasionally it reduces Benedict's or Fehling's solution. The scanty urine is the result of desiccation of the blood. The kidney becomes functionally inactive, although usually no demonstrable abnormalities are found in the kidney at autopsy. This alteration in the functional capacity of the kidney results in the accumulation in the blood of products ordinarily eliminated by the urine.

The total nonprotein nitrogen of the blood increases and may be as high as in cases of uremic coma. Amounts above 200 mg. for each 100 ml. are not uncommon. The blood chlorides are increased in most cases of severe anhydremia and may be very high.

In most cases of anhydremia the blood bicarbonate is reduced, sometimes to very low levels. We have not infrequently observed cases in which the volume percentage of carbon dioxide had fallen lower than 10 volumes per cent (4.5 milliequivalents to the liter) indicating a reduction of bicarbonate to one-fifth of the normal. The bicarbonate reduction is associated with the development of all of the symptoms and associated findings of acidosis.

Remarkable alterations in the character of the respirations may occur. The respirations, instead of being chiefly abdominal, as in the normal infant, become both costal and abdominal. The whole thorax rises with each inspiration and the accessory muscles are brought into play. The breathing is deep, pauseless, but not especially rapid. It is the same "air hunger" type of breathing as is seen in diabetic coma, and is indicative of acidosis.

Some degree of fever is usually present; the temperature may be high in severe anhydremia. In some instances the fever is due to infection; in others, however, it seems to be due to a disturbance of the heat-regulating mechanism as the result of an insufficient amount of water in the body, and may subside when sufficient fluid has been administered.

The mental condition of these babies is at first one of restlessness and excitement. Later, if the condition is untreated, the infant frequently lapses into a state of coma. Convulsions are not infrequent and often close the scene. Collapse symptoms may occur at any time. Vomiting often occurs as the result of anhydremia; it is seen in cases in which the anhydremia is the result merely of a diminished fluid intake and resembles the vomiting that occurs in animals fed exclusively on solid food. The vomiting often ceases when the water content of the body once more becomes normal.

Acidosis

When diarrhea is severe, and especially if associated with a considerable degree of anhydremia, acidosis is likely to develop. The acidosis is the result of a combination of factors. The fluid present in diarrheal stools represents in part unabsorbed gastrointestinal secretions. These secretions contain mineral salts. In the gastric juice, acid ions predominate; in the duodenal and intestinal secretions, basic ions; in the combined total secretion of the gastrointestinal tract, bases predominate over acids in about the proportion of 3:2. Failure to absorb the secretions from the gastrointestinal tract results therefore in a continuous depletion of the base or alkali of the body.

In the presence of anhydremia, secretion of urine is greatly diminished, so that less acid is removed from the body and a concentration of acid metabolites in the blood occurs. Furthermore, one of the mechanisms for compensating against acidosis, namely neutralization of acids by ammonium salts, becomes ineffective when on account of diminished renal function the ammonium salts cannot be formed and eliminated. One result of anhydremia is to diminish the blood volume and thus to impair the circulation. With poor circulation and consequent anoxemia, some accumulation of lactic acid occurs in the blood and this serves still further to neutralize the bicar-

bonate present. The partial starvation occurring due to decreased food intake and absorption occasionally results in some overproduction of the ketone acids, beta-oxybutyric and acetoacetic, although ketosis is usually not a very important factor in contributing to the acidosis associated with diarrhea.

While acidosis is the expected result of severe diarrhea, several observers have reported the occurrence of alkalosis. The condition is rare and is produced by some intrinsic functional defect by which chloride is excreted in the stool in excess of sodium. Sodium excretion in the stools is increased, but not so much as chloride excretion. The stool is more acid than the urine, and little chloride is excreted in the urine. In correction of the condition ammonium chloride did not prove as useful as sodium chloride.

Toxemia From Intestinal Bacteria

In diarrhea of the infectious type, or in bacillary dysentery, the toxemia of the infection may in itself be sufficient to bring about a fatal outcome. Some infants succumb to toxemia even before very severe diarrhea has developed.

The Differential Diagnosis of Diarrhea

Inasmuch as diarrhea is caused by a variety of conditions and as the treatment to be adopted depends, to a considerable extent, upon the character of the diarrhea, an effort should be made, in each case, to determine if possible the underlying cause.

A careful case history is essential in arriving at a diagnosis as to the type of diarrhea. It is necessary to know the character of the previous feeding, whether the food mixture has been of suitable composition or whether any constituents have been deficient or present in excess; whether the feedings have been given at proper intervals and, in the case of artificially fed babies, whether the formula has been prepared from raw or heat-treated milk;

whether articles of diet other than milk have been given, and whether or not diarrhea has been epidemic in the neighborhood. It is also important to know whether the infant has shown any symptoms of infection outside of the gastrointestinal tract, such as, for example, a cold in the head or running ears.

If the food has been of suitable composition, and if it has been fed at proper intervals and has previously agreed with the infant, it may safely be assumed that the diarrhea is due to some extraneous factor, most likely a parenteral infection. Such an infection should particularly be suspected if fever and vomiting have preceded the onset of diarrhea. In order to confirm the diagnosis, a careful search should be made for the presence of infection. The nose, throat, and ears, especially, should be carefully inspected. The urine should be examined microscopically for the presence of pus. Ordinarily, however, pyelitis is less likely to lead to diarrhea than is either rhinopharyngitis or otitis media. If high temperature is coincident with the development of diarrhea, and if no parenteral infection is discovered, one may suspect the presence of bacillary dysentery, but an absolute diagnosis cannot always be made early in the course of this disease. Later, the appearance of blood and pus in the stools, or the finding of dysentery bacilli on stool culture, may make the diagnosis clear. Diarrhea caused by virus infection is diagnosed by exclusion of other causes, by its persistence despite treatment, and often by history of exposure in an epidemic.

Diarrhea due to underfeeding is, of course, seen only in infants who have been grossly underfed, especially those who have reached a state of undernutrition. The stools of hunger diarrhea are usually not large, although they may be numerous. They are likely to be dark-colored and do not contain much in the way of food remnants.

The diarrhea occurring in infants suffering from gastroenterospasm is chronic, except in those cases due to acute

infection, and is accompanied by other evidences of autonomic imbalance, such as colic, vomiting, and a general tendency to hypertonicity.

Diarrhea due to overfeeding can best be diagnosed from the history of the giving of an unsuitable milk mixture in which some one food component has been present in large excess. Examination of the stools gives but little information as to the original cause of the diarrhea. An infant who has been receiving any fat in the food is likely to show some neutral fat, soaps, or fatty acids in the stools; likewise, when diarrhea develops in an infant receiving any carbohydrate, the stools are likely to be acid in reaction, irrespective of the original cause of the diarrhea. The appearance of starch in the stools indicates merely that starch has been fed and that some has escaped digestion. No significance can be placed upon this finding in young infants, since they do not normally digest starch.

Bacteriologic examination of the stools is of value in differentiating the diarrhea due to enteric infections from other types. In the diarrheas due to parenteral infections or to unbalanced diets, the bacteriology of the stools does not differ from that of the normal infant. When enteric infections have occurred, the offending organism may be found on culture of the stools. The finding of organisms of the dysentery group establishes the diagnosis of bacillary dysentery. Besides the "true" dysentery bacillus of Shiga, a large group of closely related organisms exist which are capable of causing enteritis. Various atypical strains of colon bacilli, a number of types of streptococci, the *B. mucosus*, the *Shigella* group of bacilli (*B. dispar*), *Pseudomonas aeruginosa* (*B. pyocyaneus*), and other organisms may occasionally lead to epidemics of diarrhea.

It is questionable whether the presence of the gas bacillus (*Clostridium perfringens*) is of significance. This organism is often found in normal stools and is sometimes pres-

ent in large numbers in diarrheal stools. It is possible that it may, in some instances, produce sufficient butyric acid from the decomposition of sugars to cause intestinal irritation. Some strains of the gas bacillus also produce histamine. Some importance has been attributed to the finding of predominantly proteolytic or predominantly fermentative types of bacteria in the stools, and efforts have been made to use these findings as a basis for treatment which consists in reducing either carbohydrates or proteins in the diet. Unfortunately, this procedure has not proved to be of great practical value because of the fact that the findings are not clear-cut. Both normal and diarrheal stools contain both types of bacteria and either may predominate. Alterations in the diet only occasionally change the predominating type of flora, and such change is by no means regularly associated with clinical improvement.

Prophylaxis of Diarrhea

Infantile diarrhea is largely preventable. Short periods of diarrhea may occur in the case of almost any infant, even with the best of care, but serious or fatal diarrhea need not occur if an infant is fed properly and lives under good hygienic conditions. Breast feeding is one of the best means of preventing severe diarrhea because there is little chance of introduction of harmful microorganisms into the intestinal tract; and because human milk is readily digestible and when fed, favors the maintenance of conditions in the gastrointestinal tract which are unfavorable to the growth of bacteria capable of producing gastrointestinal disturbance. Furthermore, breast-fed infants seem less susceptible to those infections that so commonly cause diarrhea in artificially fed babies.

In the case of the artificially fed infant, absence of harmful bacteria in the milk is most important. The general improvement in the market milk supply and the almost universal custom of boiling milk for infant feeding have been important factors in the reduction of mortality from

diarrheal diseases. Boiling of milk not only reduces the bacterial content, but also renders the milk more readily digestible.

Also of importance for the prevention of diarrhea is the construction of a milk formula which is readily digestible and which does not approach too closely, in any particular, the digestive tolerance of the infant. Methods for constructing formulas are discussed fully elsewhere. A factor of greatest importance in producing ready digestibility is the treatment of the milk in some such manner that a fine curd is produced either before or after ingestion. A factor of some significance in the prevention of diarrhea is the acidification of milk. Appropriate addition of acid produces a fine curd and decreases buffer capacity. Acidification is not necessary for the normal infant, but lowered buffer capacity is of value when gastric secretions are decreased because of illness or malnutrition.

The proportion of sugar added to the formula is often of considerable importance in producing or avoiding diarrhea. Most young babies will have loose stools if the amount of sugar added is 10 per cent or more of the total formula—or of the amount of *milk* present, in the case of formulas with little dilution. Additions of 5 to 6 per cent are much safer, and with such additions the energy requirement may be met fully. When the proportions of constituents in a formula are appropriate and of a magnitude discussed in the chapter on artificial feeding, much less danger of diarrhea exists, even when the total amount fed exceeds the energy requirement considerably.

A sufficient intake of water, especially during the warmer months of the year, is an important factor in the prophylaxis of diarrhea.

Overheating of the body results in decreased digestive capacity, which is likely to result in diarrhea. Infants should not be overclothed or kept in hot, humid rooms, but they can stand considerable summer heat if kept out-of-

doors or in well-ventilated quarters, and if given plenty of water and not overclothed.

Maintenance of the nutrition is one of the important means of preventing diarrhea. The undernourished infant is especially likely to suffer from diarrhea and from the infections which lead to diarrhea. Many more infants have died from diarrhea which, in the ultimate analysis, has been the result of underfeeding than have died from overfeeding.

Diarrhea occurring in infants who have been fed properly is in most instances the result of infection somewhere in the body. This common type of diarrhea may be prevented by prompt recognition and suitable treatment of infections as they occur.

Treatment of Diarrhea

It is not always possible to determine at the outset whether the diarrhea will be of a mild or severe type; hence it is safer to consider all diarrhea as potentially severe and to treat the infants accordingly. A day or two of proper treatment early in the course of diarrhea may be far more effective than a week or two of treatment later, after a period of neglect.

General Principles

Certain general principles of treatment apply to all forms of diarrhea. These are:

1. Recognition and suitable treatment of parenteral infections.
2. Rest of the gastrointestinal tract.
3. The giving of food adapted to the limited digestive capacity.
4. Restoration and maintenance of the fluid balance.
5. Restoration and maintenance of the mineral balance.
6. Blood transfusion in cases of severe diarrhea.

In the treatment of any infant with diarrhea, the first step should be to determine, if possible, the underlying

cause. A careful search for any form of parenteral infection should be made. More information is often obtained from examining the ears of an infant suffering from diarrhea than from examining the stools. In those instances in which diarrhea is caused by parenteral infection, the results from treatment are unsatisfactory so long as the infection is present.

In any form of diarrhea, the digestive function is impaired and the intestinal tract is either irritated or is the seat of abnormal bacterial infection. In any event, food is imperfectly digested and absorbed, and any unabsorbed excess may be decomposed and cause further irritation. An excess of food not only serves no good purpose but is a source of potential harm. In all cases of diarrhea, it is desirable that the intestinal tract be given a rest. The only fully satisfactory method of accomplishing such rest is the withholding of all food for a period of time, during which only water is given. The duration of the period of starvation will depend on the age and nutritional condition of the infant, the severity of the diarrhea, and the reaction to starvation. A single starvation period at the onset of diarrhea is preferable to repeated periods of starvation or to prolonged underfeeding. In bacillary dysentery, and in many of the diarrheas due to parenteral infection, starvation, even if prolonged, is likely to have but little effect in causing a cessation of the diarrhea.

After the starvation period, the food given an infant with diarrhea should be nonirritating, readily digestible, and of a type which tends to bring about normal chemical conditions in the gastrointestinal tract. A relatively low fat content is desirable, inasmuch as fat tends to delay the emptying of the stomach and predisposes to vomiting; hence skimmed or partially skimmed milk is preferable to whole milk. Such milk as is used should be so treated that only very fine curds are produced during the process of gastric digestion. Boiled, evaporated, dried,

or acidified skimmed milk meets these indications. The formula should be of such character that, when fed, conditions are produced in the gastrointestinal tract which are unfavorable for bacterial action. Inasmuch as the sugars in general often serve to aggravate diarrhea, only moderate amounts should be given, and the carbohydrate used should be selected on the basis of least laxative effect. Thus dextrose, dextrin, and dextrin-maltose mixtures are preferable to lactose and sucrose.

Foods which meet the above requirements and which have proved especially serviceable in the feeding of infants with diarrhea, are acidified skimmed (or partially skimmed) boiled fresh milk, dried skimmed milk, and protein milk. To these various forms of milk, moderate amounts of maltose-dextrin preparations, or of dextrose, are added. Dextrose is very quickly absorbed, and when given in a concentration of less than 6 or 7 per cent is not irritating to the intestinal mucosa. The parenteral administration of dextrose solution may be resorted to as a means of introducing extra food.

In all forms of diarrhea, it is essential that a sufficient amount of fluid be given to maintain a normal water balance of the body. Unless body fluids can be maintained, other methods of treatment are likely to fail. The amount of water which may be taken by mouth is often limited because of persistent vomiting. A larger total amount of water often can be taken when the water is given in small amounts at short intervals than when larger amounts are given less frequently. When it is impossible to administer a sufficient amount of fluid by way of the gastrointestinal tract to prevent the symptoms of dehydration, recourse must be had to parenteral means of fluid administration. Fluid may be given subcutaneously, intravenously, or intraperitoneally. Intravenous administration restores more quickly a depleted blood volume and is indicated in all cases of acute anhydremia accompanying diarrhea. By the use of the subcutaneous and intraperitoneal routes, much

larger quantities of fluid may be introduced than intravenously—unless the intravenous administration be continued for several hours.

For intravenous injection, dextrose solutions of a strength of from 10 to 20 per cent may be used and such solutions may be combined with various salt solutions: salt or Ringer's solution, or Hartmann's physiological buffer salt solution. Saline solutions may be given intravenously, subcutaneously, or intraperitoneally. Ordinary physiological sodium chloride and Ringer's solution are suitable for the treatment of mild degrees of dehydration accompanying diarrhea. In cases of more severe diarrhea, however, a preferable solution is Hartmann's physiological buffer salt solution or Darrow's buffered potassium chloride solution. (See Chapter XXXII.)

Inasmuch as in all cases of severe diarrhea a loss of mineral results in which the fixed bases predominate, restoration of the mineral content of the body is essential. Such restoration may to some extent be accomplished by means of the food, but is more quickly accomplished, especially in severe types of diarrhea, by the parenteral administration of salt solutions designed to restore the depleted elements.

In the severer types of diarrhea, repeated transfusions with citrated whole blood are of the greatest benefit. Transfusions improve the circulation and lead to improvement in the digestion and absorption of food. The injected blood also supplies materials for the reconstruction of damaged body cells and may have some effect in increasing the infant's resistance to infection. Transfusions should not be given, however, until the fluid balance of the body has been restored.

Treatment of Diarrhea in the Breast-Fed Infant

In infants fed exclusively at the breast, diarrhea is rarely severe or of long duration. A moderate increase in the number of stools and a slight change in their character,

if unaccompanied by fever or constitutional symptoms, is of only slight significance. The only treatment indicated, in such instances, is temporary reduction in the food intake, which may be accomplished by lengthening the feeding intervals (if less than four hours) and shortening the time of each nursing.

When the diarrhea is more severe, and especially if fever is present, a search should be made for parenteral infections, since these are the most common causes of diarrhea in breast-fed infants. Diarrhea is not an indication for weaning, although the food intake should be diminished for a period. At the start, several feedings may be omitted and the infant given water or barely water, which may be sweetened with saccharin (15 mg. to 125 ml. or $\frac{1}{4}$ gr. to 4 oz.) if desired. If the diarrhea is severe, starvation should be continued for as long as 24 hours, a period amply long for breast-fed infants. During the period of starvation, the mother's breasts should be emptied by manual or mechanical expression. When the feedings are resumed, the intervals should not be shorter than every four hours, and the infant should suckle for only a few minutes. It is advisable to offer several ounces of water previous to putting the infant to the breast. If water is offered from a bottle just preceding the nursing, the infant is not likely to suckle as vigorously or to take as much milk, and such milk as is taken will have a low fat content as the breast is incompletely emptied. Supplemental foods should be omitted during the period of acute diarrhea. In most instances, diarrhea of breast-fed infants responds to the simple treatment of temporary withdrawal of food followed by a diminished food intake. In some instances, recovery is hastened by the administration of a buffered lactic acid solution (see p. 299) preceding each nursing. From 30 to 90 ml. (1 to 3 ounces) of this solution may be given. Small feedings of boiled skimmed milk, skimmed lactic acid milk, or protein milk, just preceding the nursing, sometimes have a beneficial effect. Some infants who

are accustomed only to feedings at the breast, may refuse any acidified foods because of the sour taste.

If the diarrhea fails to respond within a reasonable time to these simple methods of treatment, the presence of unrecognized parenteral infection should certainly be suspected. In rare instances a continuation of the symptoms is due to enteral infection.

The Treatment of Mild Diarrhea in Artificially Fed Infants

In well-nourished artificially fed infants, and especially in those over four or five months of age, diarrhea is usually mild in type and responds promptly to suitable treatment. In very young infants, in those who have been fed inadequately and improperly, and in infants who have suffered from chronic or repeated acute infections, diarrhea is likely to be much more severe.

The first step in the treatment should be to ascertain, if possible, the cause of the diarrhea. A search should be made for infections, which, if found, should be treated appropriately. If such an acute infection as otitis media is discovered and the eardrum is opened, there may be little need for further treatment. If the food has been appropriate, the regular feedings may be continued but in smaller amounts for a few days. In those cases in which the infant has been receiving a sweet-milk formula, it is often advantageous to resort, at least temporarily, to a formula prepared from acidified milk. It is advantageous to reduce the sugar content somewhat and such sugar as is used should be either dextrose or of the maltose-dextrin type.

If no source of infection can be found, or if such infection as is found is not amenable to immediate treatment, a more strict regulation of the diet is necessary. A period of starvation should be instituted, during which the infant receives an abundant amount of water. The length of the starvation period will depend on the age and nutritional

condition of the infant, as well as on the severity of the diarrhea. The omission of one or two feedings often suffices. During the starvation period, a buffered lactic acid solution may be given. (See p. 299.)

A method of treatment often employed, especially for older infants, consists in the feeding of finely scraped raw apple, which is given at two- or three-hour intervals in amounts of one to four tablespoonfuls at a time. A total of as much as 1000 to 1500 grams of the apple pulp is given in the twenty-four hours; the pulp comprises the sole nourishment for two or three days. No question exists as to the effectiveness of the apple diet. The effects are attributed chiefly to pectin, through its assumed ability to remove toxic substances because of its colloidal character and through its being a source of galacturonic acid, a detoxifying agent, in the intestine. Canned apple powder is available commercially, and is as useful as scraped raw apple and much more convenient. Pectin alone, or in combination with agar, or with agar and a dextrin-maltose preparation, is frequently used instead of apple. A commercial product containing approximately 5 per cent pectin, primarily intended for the preparation of fruit jelly, is available everywhere. The dosage of this preparation varies from 60 to 180 ml. (2 to 6 ounces) daily, depending on the age or size of the infant or child. At least one commercial firm markets a mixture of pectin, agar, and a dextrin-maltose preparation, intended especially for the treatment of babies with diarrhea. For babies with mild diarrhea, not only is a fasting period unnecessary as a preliminary to the pectin treatment, but a modified milk formula containing the pectin may be fed. In cases of more severe diarrhea, the use of pectin without milk seems preferable in the beginning.

After a short period of starvation, or of apple or pectin diet, the feedings may be resumed. In the case of older infants unaccustomed to the taste of acid milk, as suitable a feeding as any is boiled skimmed milk without added

sugar. The amount given at first should not be more than about one-half or two-thirds the usual volume taken at a feeding by the infant. After a few feedings, the amount of milk given may be increased, and if the diarrhea shows signs of cessation, a moderate amount of carbohydrate is added. The proportions should not be greater than one part of sugar to twenty of milk. If all goes well, whole milk may be substituted gradually for the skimmed milk in the formula. Additional carbohydrate may then be added until the infant is receiving the usual formula for the age.

In infants who have been taking acid-milk mixtures, the only changes in the diet indicated in the presence of mild diarrhea are a reduction in the sugar content and a moderate reduction in the total volume of food taken, the difference being made up with water. If the diarrhea does not respond promptly to this form of treatment, or if there are constitutional symptoms present, acid skimmed milk or protein milk should be used, as in the treatment of the severe types of diarrhea (see p. 306). The decision as to how rapidly the food intake may be increased will depend more on the general constitutional symptoms than on the character of the stools. The infant should be carefully watched for the appearance of any of the symptoms of intoxication, especially fever, desiccation, grayness of the skin, and the nervous manifestations. Any sudden or marked loss of weight is of great significance. When any of these symptoms are present, the diet should be increased only with the greatest caution, and in addition other methods of treatment, such as the administration of fluids and transfusion, are indicated.

In the presence of diarrhea, the intestinal tract usually empties itself well without the aid of cathartics, but if the infant is known to have taken an unsuitable article of diet, or if the diarrhea has barely started, a cathartic may be administered in order to remove the offending material before damage has occurred. Usually, however, the infant is seen after the diarrhea has continued for some time and

no good is to be accomplished through the use of a cathartic. Certainly not more than one dose should be administered in any event. If a cathartic is used at all, castor oil is as satisfactory as any. A suitable dose is from one to two teaspoonfuls, depending on the size of the infant. Calomel should not be used, as it possesses no advantages over other cathartics and may cause severe irritation of the gastrointestinal tract.

Treatment of the Severe Forms of Diarrhea

(ALIMENTARY INTOXICATION, TOXICOSIS, CHOLERA INFANTUM, ANHYDREMIA)

Diarrhea of a very severe type is seen in badly undernourished infants, especially those who have been artificially fed and in those who have suffered from chronic or repeated acute infections. The diarrhea is accompanied by symptoms of marked toxicity, fever, and dehydration. The prognosis is grave and the treatment must be energetic. Fortunately, except for epidemic diarrhea of the newborn, these severe types of diarrhea are less frequent than in previous years, and occur only rarely in private practice or among infants who have been under careful medical supervision. At the outset such diarrhea may be indistinguishable from an ordinary diarrhea, but severe toxic manifestations soon make their appearance. The temperature rises, the appearance of the infant changes, the skin becomes gray in color and loses its turgor. The eyes are sunken and have a far-away, glassy stare. All the symptoms of anhydremia are present. Vomiting and diarrhea become severe, the stools increase in volume and they finally consist of little more than brownish colored water. As the condition develops, acidosis occurs and the infant's respirations become deep and pauseless.

In beginning treatment, an effort should be made to locate any causative infection, and if any is found it should be appropriately treated, but there should be no delay in restoring lost body fluids and mineral salts.

Water is urgently needed and as much as possible should be given by mouth. Unfortunately the persistent vomiting often precludes the possibility of giving very much fluid in this way. Small amounts of water at short intervals are often better retained than larger amounts given less often. The total amount of fluid required daily in the presence of acute dehydration may be over a quart (1000 to 1500 ml.), and it is only under the rarest conditions that all of this can be given by mouth. If fluid is retained when given by mouth, it is well to give in addition to water some mineral salts and potential alkali. The water given may be mixed with from one-third to one-fourth of its volume of Ringer's solution. This supplies sodium, potassium, and calcium salts, and chlorides. Additional potassium may be needed when dehydration has been marked; it may be given as Darrow's solution. Potential alkali may be given by mouth in the form of sodium lactate solution. Sodium lactate, although neutral in reaction, is converted into sodium bicarbonate after being absorbed, and supplies necessary base to overcome the acidosis which is usually present. A solution of lactic acid buffered with sodium lactate serves a double purpose: the sodium lactate provides base and the lactic acid, through bringing about a condition of acidity in the upper intestinal tract, inhibits the growth of bacteria. A solution of this type may be prepared as follows (Formula of Dr. Alexis F. Hartmann, but not to be confused with Hartmann's physiological buffer salt solution):

Buffered Lactic Acid Solution for Oral Use

Lactic acid U.S.P.	15 ml.
Sodium hydroxide 10 per cent	20 ml.
Water to	1000 ml.

The solution may be conveniently prepared in concentrated form of ten times this strength, and diluted 1:10 with water before use. The buffered solution is given in addition to water by mouth. The solution may be given

alone in amounts of from 25 to 100 ml. (one to three ounces) at two- or three-hour intervals (if the infant can retain it), or in smaller amounts at shorter intervals; or it may be added to the water given. An effort should be made to give from 300 to 600 ml. from (10 to 20 ounces) in the course of twenty-four hours.

The administration of fluids by mouth must, in most instances, be supplemented by the injection of fluids parenterally. In many cases of severe diarrhea it may not be possible to administer any fluid by mouth, so that the entire fluid requirement must be met in other ways. Fluids may be given intravenously, subcutaneously, or intraperitoneally, or by a combination of these methods. The choice of fluids for parenteral administration lies between physiological salt solution, Ringer's solution, sodium lactate, dextrose, Darrow's buffered potassium chloride solution, and combinations of these. Isotonic sodium chloride and Ringer's solution actually contain a higher percentage of sodium chloride than the blood and tissue fluids and, in some cases of severe dehydration with chloride retention, the injection of these solutions may result in an increase in the blood chloride and a corresponding decrease in blood bicarbonate and thus lead to an increase in the degree of acidosis. When dehydration is not severe, the injection of a sufficient amount of these isotonic solutions may result in the establishment of urinary secretion and the excretion of retained sodium chloride or other metabolites. Neither of these solutions, however, supplies alkali, which is required when any degree of acidosis is present.

Alkali may be supplied parenterally in the form of sodium lactate. A solution of one-sixth molar* strength is isotonic and nonirritating and may be injected intravenously, subcutaneously, or intraperitoneally. After injection, the lactate is converted into sodium bicarbonate and in this way restores depleted alkali and overcomes the

*Molar sodium lactate may be obtained in sealed ampules. To prepare the one-sixth molar solution, the contents of the ampules are diluted with five times the volume of sterile distilled water.

existing acidosis. The injection of 60 ml. of one-sixth molar sodium lactate for each kilogram of body weight is sufficient to raise the blood carbon dioxide by 30 to 35 volumes per cent (15 to 16 milliequivalents to the liter). This amount represents the maximum usually injected at one time in the beginning of treatment when severe acidosis is present, as indicated by the symptoms or by blood analysis. When the acidosis is less marked, smaller amounts of the lactate solution are necessary. Of the total amount of sodium lactate solution given, one-third may be introduced intravenously and two-thirds subcutaneously or intraperitoneally as the first step in treatment.

After these initial measures for the overcoming of such acidosis as may be present, fluid administration is continued, either by a combination of subcutaneous or intraperitoneal injection, or by a continuous intravenous injection (venoclysis). For the subcutaneous or intraperitoneal injections, the solution of choice is a combination of Ringer's solution with sodium lactate of such a strength as to be slightly hypotonic. Such a combined solution is known as Hartmann's solution (physiological buffer salt solution*).

For the intraperitoneal injections, as large an amount of solution is given as the abdominal cavity will comfortably hold. The injections are repeated at intervals of from six to twenty-four hours, depending on the rate of disappearance of the injected fluid and the evidences of restoration of body fluid as shown by a disappearance of the manifestations of anhydremia—improved color and turgor

*This solution may be obtained in concentrated form in ampules and diluted before use. The formula follows.

HARTMANN'S SOLUTION FOR INTRAVENOUS USE

Stock solution to be diluted 25 times before use. All reagents should be C. P. quality.

Lactic acid, 85 per cent solution	60 ml.
NaCl	150 Gm.
KCl	10 Gm.
CaCl ₂ ·H ₂ O (or anhydrous CaCl ₂ 4.3 Gm.)	5 Gm.
Powdered phenol red for indicator.	
Saturated NaOH solution, CO ₂ free, to neutralize.	
H ₂ O, freshly distilled, sufficient to make	1000 ml.

Boil for 30 minutes to convert the lactic acid anhydride present to lactic acid. Re-neutralize with saturated NaOH as often as the solution becomes acid. Cool and bring back to the original volume with distilled water. Filter, measure into test tubes, autoclave at 15 pounds for 30 minutes and seal.

of the skin, beginning secretion of the urine, and increase and maintenance of body weight. The same solution may be given subcutaneously instead of intraperitoneally. It should be borne in mind that infants suffering from severe diarrhea and vomiting are continuously losing fluid and salts so that even though the body fluids are restored, anhydremia and acidosis persist unless repeated injections are given. In some cases it is necessary to continue the daily parenteral administration of fluid for a period of weeks. One small infant under our care was given seventy-two intraperitoneal injections over the course of several weeks, the total volume of injected fluid being over ten gallons (40 liters)! Ultimate recovery followed.

Another very effective method of fluid administration, although somewhat more difficult technically, is that of continuous intravenous injection (venoclysis). One advantage of this method of fluid administration is that nothing whatsoever need be given by mouth during the period that venoclysis is continued. The technique of venoclysis and the solutions to be used are considered in Chapter XXXII.

When continuous venoclysis is not employed, intravenous injections of dextrose may be combined with the intraperitoneal or subcutaneous injections of saline solutions. Dextrose solutions having a strength of 10 to 20 per cent may be given daily, or twice daily, in amounts not exceeding 25 ml. for each kilogram of body weight at each injection. The injection should be given very slowly: at least 20 minutes should be consumed in the injection of the total volume of fluid.

For fluid administration in diarrhea the conventional procedure has been to use physiological salt solution, Ringer's solution, lactate solution, dextrose solution and combinations of these. Darrow and co-workers have proposed an additional type of fluid, the proposal being based on studies of salt metabolism of infants with diarrhea. It was found that potassium is lost from the body

in amounts up to one-fourth the total estimated body content. The loss of potassium is not accompanied by an equivalent loss of nitrogen, indicating that cells are not destroyed, but that a transfer of potassium from intracellular to extracellular fluid and thence to the intestinal tract has occurred. The loss of potassium from the body can result seriously. Sometimes the use of the conventional fluids aggravates the potassium loss. In such instances sodium enters the cells by transfer from extracellular to intracellular fluid. Such transfer increases acidosis because chloride ions remain in the extracellular fluid. The condition of the infant is made worse.

For parenteral replacement therapy either of the following solutions is suggested by Darrow:

1. 40 ml. molar sodium lactate (one ampule)
2 Gm. potassium chloride
3 Gm. sodium chloride
710 ml. sterile distilled water
2. 2 Gm. potassium chloride
3 Gm. sodium chloride
250 ml. one-sixth molar lactate
500 ml. water

The solutions can be sterilized by autoclaving, or the chlorides can be sterilized by dry heat and added to the sterile water along with an ampule of molar sodium lactate. Up to 80 milliliters of these solutions may be given subcutaneously for each kilogram of body weight over a period of eight to twelve hours. Repeated administration may be desirable so long as fluids are needed parenterally.

In moribund and severely ill babies the concentration of potassium in the plasma may be high, despite a deficit in intracellular potassium. For such infants solutions containing potassium are dangerous and are contraindicated until urine formation is established. Hartmann's solution or saline together with glucose solution are the solutions of choice for such infants.

As soon as some food may be taken by mouth, one or two grams of potassium chloride may be added to the food mixture and parenteral potassium therapy may be discontinued. When full oral feeding is established, potassium therapy is discontinued.

Potassium intoxication is avoided by careful regulation of the dosage usually below 80 ml. for each kilogram daily, with 100 ml. for each kilogram as the ultimate maximum; by slow rate of injection, never under four hours and preferably eight to twelve hours; the solutions should never be given in shock or when urine secretion is very scanty. If diarrhea recurs a short time after potassium therapy is discontinued, no more potassium should be given without knowledge of the potassium content of the plasma. Sufficient potassium may have accumulated in body fluids to lead to heart block on introduction of further amounts. In such an event calcium is administered intravenously together with hypertonic glucose solution. In order to control the therapy and to prevent intoxication it is desirable to determine the potassium content of the plasma from time to time during the treatment.

Use of potassium therapy does not decrease the duration of illness, but it decreases mortality importantly. Darrow and co-workers report decrease in mortality rate from 29 per cent in a conventionally treated group of fifty-nine infants to 6 per cent of fifty-two infants given fluids containing potassium.

The beneficial effects of fluid administration are likely to be more lasting if this treatment is combined with blood transfusion. Transfusion serves to restore the blood volume and to maintain it. It is possible that transfusion also results in increasing the infant's resistance to infection through supplying immune bodies.

A transfusion should never be attempted until after fluid restoration has been accomplished. A transfusion in the stage of acute anhydremia may result in further concentration of the blood and exacerbation of all the symptoms.

The optimum time for the first transfusion is from 12 to 24 hours after the initial fluid injection. Repeated small transfusions are better than a single large one. The amount given at each transfusion should be from 20 to 30 ml. of blood for each kilogram of body weight. Citrated blood is used and a sufficient amount is obtained from the donor at one time for two or three transfusions. The blood may be kept in an ice box safely for as long as three or four days before being used. (For technique of blood matching and transfusion, see Chapter XXXII.) Three or four transfusions are usually sufficient, although subsequent developments may be such that additional transfusions are required later.

A period of starvation should always be instituted in cases of severe diarrhea with toxic manifestations. During this period water, saline, or buffered lactic acid solution may be given by mouth if they can be retained. When buffered lactic acid solution is being taken, it is also safe to add some dextrose to the solution. The concentration should, however, not usually be greater than 5 per cent, and never more than 10 per cent. In such concentration dextrose is not laxative, and in the presence of acid is not attacked by intestinal bacteria before it can be absorbed. The length of the period of starvation will depend on the severity of the symptoms. The general condition of the infant is a better guide as to the time when food may be begun than the character of the stools. So long as the infant is gray, anhydremic, and apathetic, has a high temperature, and appears generally "toxic," food must be withheld. It is usually necessary to continue the period of starvation for at least twenty-four hours, and this period may have to be prolonged in some instances to four or five days.

When feedings are started, the amount should at first be small, not more than 25 to 30 ml. (one ounce), and increases should be made slowly, about 15 ml. (one-half ounce) at a time, depending on the reaction of the infant to food. Too

prolonged starvation or underfeeding, in infants already badly undernourished, may be fatal; but on the other hand, too rapid increases in food may cause an exacerbation of all the symptoms and require another period of starvation, with disastrous results. The effects of starvation may be considerably minimized if dextrose is given intravenously. Amino acid solution also is useful.

The feedings are given not more often than every three or four hours. Several types of food may be used with about equal success. A satisfactory feeding consists of dried protein milk made up in buffered lactic acid solution. Some carbohydrate is added, especially in the case of very young infants, as otherwise such infants when fed protein milk alone tend to go into a state of collapse. The first formula may be prepared as follows:

Dried protein milk	30 Gm. (1 oz.)
Dextrose	15 Gm. ($\frac{1}{2}$ oz.)
Buffered lactic acid solution	300 ml. (10 oz.)

The buffered lactic acid solution is that described on p. 299.

Another and similar type of feeding consists of acidified skimmed milk, begun cautiously and without added sugar. After the first day, a 5 or 6 per cent additon of dextrose may be made.

Either of these feedings is gradually increased in volume until, without recurrence of severe symptoms, a volume normal for the age is being ingested. A gradual transition is then made to a regular maintenance diet. Scraped apple, apple powder, or pectin in one of its suitable commercial forms, may be used in conjunction with the early feedings.

There is one type of severe diarrhea which fails to respond to the dietetic treatment outlined above. This particular variety of diarrhea appears to be due to invasion of the intestinal tract with organisms capable of decomposing protein with the production of toxic products. This

is a relatively rare type of diarrhea, but is sometimes seen in epidemic form. This particular type of diarrhea must have been more frequent in the past than it is at present. It is discussed at length in many of the older textbooks, but is referred to only occasionally in the more recent literature. We have only once observed such an epidemic. It has not been determined just what organism is responsible, but it is not one of the ordinary forms of dysentery bacilli. At the outset, it is difficult or impossible to distinguish this from any other form of diarrhea. Fever, dehydration and toxic manifestations are marked. Convulsions are of more frequent occurrence than in other types of diarrhea. The stools are more likely to be foul in odor rather than sour. Blood and pus are not observed in the stools. The giving of milk in any form to these patients, even though it be acid milk or protein milk in small quantities, results in an exacerbation of all the symptoms, and if the usual methods of treatment are followed, the mortality is high. These patients tolerate carbohydrate fairly well, and indeed seem to be benefited by reasonable amounts of carbohydrate.

The treatment of diarrhea of this type should be the same as that of the other severe forms of diarrhea except for the character of the feedings. After the initial period of starvation, feedings are begun with an 8 to 10 per cent solution of dextrose or a dextrin-maltose preparation. Later, arrowroot or cornstarch gruels are given. No milk of any sort is allowed until the temperature has fallen and the symptoms of toxemia have disappeared. It may be a week or ten days before it is safe to add milk, or other protein-containing food. Some form of acid milk may then be added to the carbohydrate diet in small amounts. If well tolerated, the quantity may be increased until finally the infant is receiving the usual diet for the age. Another type of food highly useful for the period of transition from the sugar to the milk-and-sugar diet is sweetened condensed

milk in a dilution of one to eight by volume. An initial cathartic appears to be of more value in these cases than in the ordinary cases of diarrhea.

In all cases a careful search should be made for any possible parenteral infections which may be the underlying cause of the severe diarrhea. Of the various types of parenteral infection, otitis media is the most frequent contributory cause. In athreptic infants the onset of otitis media is often insidious, and severe diarrhea may appear before any local manifestations of ear infection are detectable. Even when an infection is present in the middle ear, the only evidences may be a lack of luster of the eardrum. There may be no bulging or redness. Not infrequently, after restoration of body fluids and after transfusion the signs of inflammation may be much more evident. If middle ear infection is detected, paracentesis may be followed by marked improvement in the gastrointestinal symptoms, but such a favorable result does not follow in all cases. The symptoms may persist even after the ears are freely opened. In a certain number of instances the infection spreads to the mastoid antrum and surrounding cells and becomes walled off so that it may become necessary to resort to antrotomy or mastoidectomy in order to clear up the infection. Usually the mastoid involvement is not obvious and may be detected only by most expert examination. A fairly constant sign is edema of the posterior-superior canal wall overlying the mastoid antrum. When such infection is present and untreated, dietetic measures may prove quite ineffective in controlling the diarrhea.

Otitis media is, of course, only one of the infections which may be followed by severe gastrointestinal disturbance. A careful search should be made for other possible infection, and if any is found, appropriate treatment should be instituted. Maxillary sinusitis is occasionally a cause of a most severe type of diarrhea.

Even though there may be no infection present at the onset of the diarrhea, secondary infections not infrequently

develop during the course of the diarrhea. Secondary infections in the ears are sometimes due to organisms of intestinal type (*E. coli*). Although such secondary infections are not the cause of the diarrhea, they complicate the picture and make the prognosis more grave. Treatment is indicated as in the case of the primary infections.

Medicinal Treatment

Drugs have relatively little place in the treatment of diarrhea, although sulfonamide and penicillin therapy may be indicated for the causative parenteral infection.

Cathartics are often used but are usually contraindicated. See further discussion on p. 297.

Opium in the form of paregoric may be used when the diarrhea is severe, prolonged, and accompanied by marked water loss. Opium also serves the purpose of relieving pain and of allowing the infant to obtain some rest. Marked abdominal distention is a contraindication to the use of opium. Paregoric may be given in small doses, at short intervals, up to the point of the physiologic effect of contraction of the pupils. Standing orders for the administration of paregoric or of any other opiate should not be given; a certain number of doses should be ordered, and the order then repeated only to meet definite indications. The initial dose of paregoric in the case of a young infant is usually 0.3 ml. (5 minims); for a larger infant 0.6 to 1.0 ml. (10 to 15 minims). The administration may be repeated every two to three hours for six or seven doses, or until the physiologic effect is obtained. After this, the dosage should be decreased.

Bismuth preparations have been much used in the treatment of diarrhea. They are supposed to act as demulcents, coating over the irritated intestinal mucosa and also to have a slight antiseptic effect. Bismuth subnitrate should not be used as the nitrate is likely to be reduced to nitrite by bacterial action and to give rise to toxic manifestations.

If bismuth is used at all, the subcarbonate is to be preferred, and the dosage should be large, 2 to 4 Gm. (30 to 60 grains) or more at a dose, suspended in water. Smaller doses have little effect in coating the very large intestinal area. We have never been impressed with the value of bismuth in the treatment of diarrhea.

Various antiseptics have been used with the idea of inhibiting bacterial growth in the intestinal tract. Most of the so-called intestinal antiseptics are entirely ineffective, for when given in sufficient amounts to exert any bacterial inhibiting action they are likely to prove toxic for the infant. The water-soluble antiseptics, such as the dyes, are quickly absorbed from the upper intestine, and can therefore exert an effect for only a very short time. The sulfonamide drugs are effective against many of the organisms of the intestinal tract, but it has not been customary to use them for diarrhea except in certain kinds of enteral infection (see Dysentery).

Atropine is of value in those instances in which the diarrhea is accompanied by marked gastrointestinal spasm and vomiting. The atropine appears to relieve the colicky pains and to decrease the amount of vomiting. The methods for the administration of atropine are discussed in Chapter XXIII. It should be noted in this connection that the administration of atropine sometimes leads to elevation of temperature.

The acute collapse occasionally occurring in the course of severe diarrhea is best treated by injections of caffeine or of epinephrin. The epinephrin may be given subcutaneously in doses of 0.06 to 0.12 ml. (1 to 2 minims), or may be added to the fluids which are given intravenously in the proportion of 1:20,000. In instances of acute collapse, masked mastoiditis is often found to have a causal relationship; the only evidence of mastoiditis may be sagging of the posterior-superior canal wall.

Phenobarbital is of value in allaying extreme restlessness and irritability. It is also sometimes of value in con-

trolling severe vomiting. The dosage is from 8 to 16 mg. ($\frac{1}{8}$ to $\frac{1}{4}$ of a grain) at three- or four-hour intervals.

Calcium salts have been used by Casparis in the treatment of patients with severely toxic diarrhea. The rationale of calcium administration is to neutralize the effects of guanidine derivatives which are at times present in increased quantities during periods of severe desiccation diarrhea. Calcium gluconate is given in 10 per cent solution intravenously, 5 ml. at an injection; or calcium gluconate in water may be given by mouth in a dosage of from 0.5 to 1 Gm. ($7\frac{1}{4}$ to 15 grains) at four- to six-hour intervals.

During the periods of severe toxicity associated with gray skin and cyanosis of the extremities oxygen inhalations are often of value. The infant may be kept continuously in an oxygen tent to advantage.

CHAPTER XXI

BACILLARY DYSENTERY

(ILEOCOLITIS, INFECTIOUS DIARRHEA)

Bacillary dysentery is an acute, specific infectious disease characterized by lesions in the intestinal tract, chiefly in the colon and lower ileum. The causative organism is the dysentery bacillus or one of the members of the dysentery group. This group includes the true dysentery bacillus of Shiga and the closely related strains of paradyentery bacilli. Among the latter are included the Flexner, Hiss-Russell, and Strong types. Several subdivisions of these strains have been described which differ from each other in fermentation reactions and serologic properties. The Flexner and Hiss-Russell types are more frequently the cause of dysentery throughout the United States than is the Shiga type.

Dysentery bacilli gain access to the body by way of the gastrointestinal tract. Contaminated milk and water are the most frequent sources of infection. Such organisms as escape the bactericidal action of the gastric juice and intestinal secretions find lodgment in the mucosa of the colon and lower ileum.

Pathology

The lesions of dysentery are found chiefly in the colon and to a lesser extent in the lower portion of the ileum. In mild dysentery there may be only a superficial hyperemia with outpouring of mucus. In cases of more advanced disease there is considerable round-cell infiltration of the mucosa and submucosa. In all cases of severe disease some superficial necrosis occurs. This may be extensive so that almost the entire mucosa of the colon is covered with a necrotic pseudomembrane, resembling that seen on the

throat in cases of diphtheria. In the majority of cases, however, the necrosis is confined to the area overlying the lymph plaques, where it usually results in localized ulcerations. The ulcers are fairly deep, with overhanging edges. Hemorrhages occur due to erosion of superficial blood vessels, but the ulcers rarely perforate. Secondary infection of the ulcerated areas with pyogenic organisms is common. The mesenteric lymph nodes draining the affected areas are usually swollen. In most cases of dysentery the intestinal lesions are of such nature that complete repair is possible, but when extensive ulceration is present, repair is likely to be slow, and in some instances chronic colitis may persist for months or years.

The liver is enlarged and fatty.

Dysentery bacilli are present in the lesions throughout the course of the disease, but except in rarest instances do not invade the blood stream. In most cases of dysentery the causative organism may be isolated from the stools through the use of suitable cultural methods. (See Chapter XXXII.) The organisms are not likely to be found during the first day or so of the disease or after the subsidence of the fever.

Specific agglutinins for the various strains of dysentery bacilli may be demonstrated in the blood serum toward the end of the first week of the disease. The presence of such agglutinins is of some diagnostic value, especially in those cases in which the clinical symptoms are not clear-cut and in which it has not been possible to isolate the organisms from the stools. The agglutination test is performed in the same manner as the Widal reaction, using strains of the several dysentery bacilli.

Bacillary dysentery when untreated is a self-limited disease in which recovery depends upon the development of immunity on the part of the body. The immunity consists in the production of antitoxins and agglutinins, and until such immunity has developed, the disease will continue irrespective of the character of the feeding.

Symptoms

In typical cases of dysentery in infants the onset is sudden, with severe prostration and high temperature. The infant may vomit a few times, but vomiting is neither a constant nor a persistent symptom. Convulsions may occur, especially in the case of young infants and those suffering from infection with the Shiga type of dysentery bacillus. The initial toxemia may be so severe that the infant succumbs within less than twelve hours, even before any marked degree of diarrhea has developed. The general condition of the patient is one of extreme prostration and apathy, closely resembling that of typhoid fever. The severe abdominal pain may result in periods of restlessness and irritability. Refusal of food and even of water is commonly observed, so that it may become necessary to feed by gavage. The abdomen is usually distended and often tender.

At about the time of the onset of fever or shortly thereafter the stools become loose. They are at first thin, watery, and contain large amounts of mucus, but do not differ greatly from those seen in other forms of diarrhea. In typical cases, by the second or third day, or at times even on the first day, blood appears in the stools either as small flakes or in sufficient amounts to color the whole stool. When the disease is in this stage the diagnosis of intussusception is sometimes confused with that of dysentery. A careful history and examination usually suffice to distinguish these two conditions. Pus is present in the stools in microscopic amounts early in the course of dysentery. Later it is present in macroscopic quantities. In some instances shreds of necrotic membrane may be seen. The characteristic stools in cases of well-developed dysentery are small and brownish-green, and may consist of little but blood, mucus and pus. They have a peculiar musty odor, resembling that of wet hay. The stools are passed fre-

quently, sometimes as often as every half-hour throughout the day. Accompanying the passage of the stools there is marked abdominal pain and tenesmus.

In convalescence the transition period from abnormal to normal stools commonly has a duration of three to seven days. In those cases of dysentery in which the course has been prolonged and extensive ulceration has occurred, the stools frequently continue to be bloody or purulent for as much as two weeks or more after the fever subsides. Because of continued indigestion the stools may not resume their normal character until weeks or months after the temperature has become normal and other symptoms of the disease have disappeared. The stools may continue to be numerous, eight to ten a day, and contain undigested food and some mucus, although pus and blood may no longer be present. In convalescence the stools may be formed and apparently normal for a day or two, and subsequently contain blood and pus, presumably the result of the breaking down of an ulcer. This change in the character of the stools may occur with very little exacerbation of the general symptoms.

The urine is scanty due to the diminished fluid intake and the loss of fluid by way of the bowel. During the height of the disease traces of albumin and a few granular casts usually are present in the urine. Acetone and diacetic and oxybutyric acids often are present in traces and sometimes in large amounts, even early in the course of the disease. Aetonuria is more frequent in dysentery than in other forms of diarrhea and is probably the result of liver damage by the toxins of the dysentery bacillus.

The blood shows a polymorphonuclear leukocytosis, the total white count being between 18,000 and 25,000. In some instances marked leukopenia occurs at the onset of the disease. This initial leukopenia may lead one to suspect the presence of typhoid fever. The differential leukocyte count by the Schilling method shows a very distinct in-

fectious picture, consisting of a "shift to the left." This is true both in those cases in which there is leukocytosis and in those in which leukopenia occurs.

The course of dysentery is variable, the average duration being from two to three weeks. During this time the fever is likely to be of a continuous type, with only occasional remissions. The fever usually is higher during the first week than subsequently. During the third week a tendency to enter convalescence is commonly observed. As convalescence begins, the temperature falls by slow and irregular lysis. The temperature may become practically normal for a day or two, and then rise again and remain elevated for a number of days. At any time during the course of the disease the temperature may be influenced by complicating infections, especially otitis media, pyelitis, or pneumonia. The course of dysentery may be greatly prolonged, especially in undernourished infants who have survived the acute period; in these the fever and all of the other symptoms may persist for as long as one or two months. In other cases, death may occur within the first twenty-four hours as the result of an overwhelming toxemia. Abortive attacks also are seen, especially in older infants and during epidemics. There may be only slight fever for two or three days; the stools may be loose but not of the characteristic dysenteric type. One might suppose that these were not cases of true dysentery, were it not for the fact that dysentery bacilli are isolated from the stools.

In all cases of severe dysentery marked impairment of the nutrition occurs, but with suitable dietetic management the degree of malnutrition may be controlled to a considerable extent.

Complications

Otitis media, pyelitis, and pneumonia are the chief complications which may occur during the course of dysentery. The otitis media often develops insidiously and may there-

fore be overlooked. There may be no symptoms referable to the ears, even though otoscopic examination reveals a red, bulging drum. Unless the ears are regularly examined, the first intimation of the presence of otitis media may be a discharge of pus from one or both ears.

Pyelitis is more frequent in very young infants and in cases in which the course of the dysentery is prolonged. The urine contains pus cells and colon bacilli; only very rarely are dysentery bacilli present in the urine.

Pneumonia, when occurring as a complication, is usually of the bronchial type, although lobar pneumonia may occur. Pneumonia is more frequent in very young and undernourished infants.

Prolapse of the rectum occurs in a fair proportion of cases of severe dysentery.

Prognosis

The course of dysentery and its prognosis have been changed greatly by the use of sulfonamide drugs. The preceding discussion is of the disease as it occurs without sulfonamide therapy and as it occurs until the baby comes under medical care and the diagnosis is made. Even with good symptomatic treatment and with expert care, the mortality in infancy is high without sulfonamide therapy. Young infants and those who are undernourished usually have less resistance to the infection than older persons, and the disease is more likely to prove fatal. In the presence of epidemics of dysentery, one may observe in a single family a rapidly fatal course in infants, a prolonged course with ultimate recovery in older children, and merely slight indisposition and mild diarrhea in the adults. Before sulfonamide therapy the average mortality rate in infancy was approximately 25 per cent. With sulfonamide therapy the mortality rate is much lower and the course of the disease much shorter. Percentage figures cannot be given

even approximately at present. Much depends on the time elapsed between the onset of the disease and the use of sulfonamides.

Treatment

Treatment of babies with dysentery includes production and maintenance of hydration, appropriate alimentation, symptomatic treatment for comfort, and chemotherapy.

As in all cases of diarrhea maintenance of water balance is essential. Many babies with dysentery refuse to take water voluntarily and it becomes necessary to administer fluid by gavage, by nasal drip, or parenterally. Blood transfusions are of value and are indicated when the course of the disease is prolonged beyond a week.

The diet should be one which is adequate to meet the nutritional requirements, but which is, at the same time, adapted to the limited digestive capacity. It should be clearly understood that the diarrhea of dysentery cannot be entirely checked by dietary means alone, for the diarrhea will continue so long as the infection is active and until the intestinal lesions have healed, irrespective of whether the infant is fed or starved. Any type of food, however, which is indigestible and irritating may aggravate the diarrhea.

Because of the limited digestive capacity of infants suffering from dysentery, acid milk in some form is preferable to sweet milk as a basis of the diet. Fat should not be present in the diet in any large amount because of the fact that fatty acids and soaps appear to be especially irritating to the ulcerated areas in the intestine. Also, fat tends to aggravate the gastric stasis that is commonly present in babies who are ill from any cause. The milk fed should, therefore, be partially or completely skimmed. An excess of laxative types of sugar should be avoided because of the irritating action on the lower bowel.

A suitable feeding for babies with dysentery is one which has for its basis acidified skimmed milk to which has

been added either dextrose or one of the dextrin-maltose preparations. A suitable proportion of carbohydrate to add is 30 Gm. to each 450 ml. (one ounce to each fifteen ounces) of acid skimmed milk. Such a formula should be fed at reasonable intervals and the infant should be allowed to take all he desires. Anorexia may be so extreme that little food is taken; in such cases the food should be administered by gavage. The total amount given at a feeding should be only slightly less than that ordinarily taken by an infant of the age under normal conditions. Starvation or prolonged underfeeding must be avoided. A continuation of the diarrhea is no contraindication to feeding; but if the feeding appears definitely to increase the diarrhea, the volumes of the feedings should be decreased, any fat present should be removed, and possibly the amount of sugar should be decreased.

In the case of infants over six months of age, the milk and sugar mixture may be supplemented by the addition of well-cooked cereal gruels prepared from barley flour, cornstarch, or arrowroot. The rougher whole-grain cereals, such as oatmeal, should not be used. Many older infants refuse to take any form of acid milk, and for these boiled skimmed milk or milk prepared from powdered skimmed milk may be substituted. Vegetables, fruit, and meat should be omitted until convalescence is established. As early as possible in the course of the disease it is desirable to have the diet "complete." Strained orange juice is well tolerated, as is also cod-liver oil after the most acute stage is passed. Thiamine is indicated and should be given in a form that does not aggravate the diarrhea. As the temperature falls and the general symptoms of dysentery disappear, a normal diet for the age may be resumed gradually.

A diet which has had considerable vogue in the treatment of dysentery has been one containing very little protein and considerable lactose. The theoretical basis of this diet

is the observed fact that dysentery bacilli do not readily ferment lactose, and produce but little toxin when grown in media low in protein. As a matter of fact, lactose, when administered in the food, is split largely into the fermentable dextrose and galactose before reaching the lower portion of the intestinal tract, and furthermore, the dysentery bacilli have invaded the mucosa and are capable of producing toxins there at the expense of body protein. In actual practice the lactose diet has not proved to be an effective one.

Apple and the various pectin preparations have some degree of usefulness in ameliorating the diarrhea of dysentery. The manner of use is discussed under Diarrhea.

Opium is of value in relieving pain and tenesmus and in slowing down intestinal peristalsis. The disadvantage in the use of opium is that it tends to increase abdominal distention. Opium may be given in the form of paregoric, beginning with an initial dose of 0.3 to 0.6 ml. (5 to 10 minims). Phenobarbital also is useful in the relief of tenesmus, pain, and restlessness. It may be used alone or in combination with opiates for this purpose. Bismuth subcarbonate in large doses may have the effect of coating the ulcerations and relieving irritation. Irrigation of the colon with dilute silver nitrate solution has been used with the idea of promoting healing of the ulcers. These irrigations are extremely painful, accomplish little good, and are not to be recommended. Irrigation of the colon with plain water or normal saline serves to remove mucus and at times appears to relieve tenesmus. It is well to follow the irrigation with a retention enema of starch paste containing from 0.2 to 0.3 ml. (3 to 5 minims) of laudanum. The use of cathartics is contraindicated in all cases of dysentery.

The administration of antidysentery sera would appear to be a logical method of treatment. If any effect is to be obtained, however, the serum should be one which is specific for the particular infecting organism. Polyvalent sera

have been prepared by immunization of horses with cultures of the various types of dysentery bacilli. The results from the use of such sera have, in general, been disappointing, although in some reported epidemics of Shiga bacillus infection, good results have been obtained when the specific serum was administered very early in the course of the disease. If serum is to be used at all, it should be given early in the disease and in doses of from 10 to 20 c.c. subcutaneously once or twice daily. It has been claimed that serum given by rectum is sometimes effective. We have had no experience with this method of administration.

The value of vaccines in the treatment of dysentery has not been demonstrated.

Chemotherapy

The sulfonamide drugs have proved of great value in the treatment of persons with bacillary dysentery. Opinion differs somewhat as to which preparation is preferable, but differences between them in effectiveness are not remarkable. Originally sulfaguanidine was used because of its poor absorption and its effectiveness within the gastrointestinal tract. Those who have used this drug extensively recommend 0.1 Gm. for each kilogram of body weight ($\frac{3}{4}$ grain for each pound) as the initial dose. One-half of this amount is then given each four hours thereafter until the number of stools daily has decreased to less than five, when the interval is increased to eight hours for a further period of two or three days or until the temperature has become normal and stool cultures are negative. Because in a small proportion of babies the dysentery is uninfluenced by this procedure, a larger dosage for all seems desirable. In fact, twice the dosage stated is within reasonable limits when dysentery is severe.

More recently preference has been shown for succinyl sulfathiazole and sulfapyrazine. The recommended initial dose is 0.25 Gm. for each kilogram ($1\frac{3}{4}$ grain for each

pound). The daily maintenance dose is the same amount divided into six doses, given at four-hour intervals.

Regardless of which drug is chosen, better results are obtained if treatment is started early in the course of the disease before the lesions in the colon have become severe. If ulceration has occurred extensively, at least moderate indigestion may be expected to continue after the causative organisms have disappeared. Decrease in fever and improvement in the appearance of the baby are observed within one or two days. At the time the fever decreases or within one or two days subsequently improvement occurs in the number and character of the stools. Some abnormality of the stools may be expected for as long as anatomical lesions remain.

CHAPTER XXII

THE CELIAC SYNDROME

The term celiac syndrome is often used to designate a group of chronic gastrointestinal disturbances characterized by the passage of bulky, foul stools, abdominal distention, and severe malnutrition due to lack of absorption of foodstuffs. Some clinicians include under this term such diverse conditions as malrotation of the bowel and tuberculous enteritis. More commonly the term celiac syndrome is used to designate intolerance for fat and starch and includes celiac disease and fibrocystic disease of the pancreas. Occasionally certain intestinal parasites, especially *Giardia lamblia*, cause symptoms similar to those of the celiac syndrome.

Celiac Disease

(CHRONIC INTESTINAL INDIGESTION, INTESTINAL INFANTILISM, IDIOPATHIC STEATORRHEA, GEE-HERTER DISEASE)

Celiac disease was first clearly differentiated by Gee, of St. Bartholomew's Hospital, in 1888. His original description was as follows:

“There is a kind of chronic indigestion which is met with in persons of all ages, yet is especially apt to affect children between one and five years old. Signs of the disease are yielded by the feces; being loose, not formed, but not watery; more bulky than the food taken would seem to account for; pale in color, as if devoid of bile; yeasty, frothy, an appearance probably due to fermentation; stinking, stench often very great, the food having undergone putrefaction rather than concoction. The pale, loose stool looks very much like oatmeal porridge or gruel. The hue is somewhat more yellow, otherwhile more drab.

“The patient wastes more in the limbs than in the face, which often remains plump until death is nigh. In the

limbs, emaciation is at first more apparent to hand than to eye, the flesh feeling soft and flabby.

“To *diarrhea alba* add emaciation and cachexia, and we have a complete picture of the disease.”

This description covers the essential features of the disease. Typical cases of celiac disease are not common. They appear to be somewhat more numerous in the southern states than elsewhere in the United States.

Etiology and Pathology

Celiac disease is not seen in breast-fed infants and is rarely seen during the first year of life. In some cases a history of improper feeding is obtained. Occasionally the disease follows an attack of diarrhea. Herter believed the condition to be due to persistence of an infantile type of flora in the intestinal tract, but the evidence in support of this view is not convincing. Because of the light color of the stools, deficiency in bile secretion has been considered by some as a causative factor. The liver is smaller than normal, but microscopically shows no changes. Pancreatic insufficiency does not occur in celiac disease, although it is an important factor in cystic fibrosis of the pancreas which is discussed subsequently.

A frequent finding in celiac disease is gastric achlorhydria. An excessive hydrolability has been observed in many of the children with this disease.

The evidence indicates that celiac disease is the result of a functional rather than an organic disturbance. Experimental observations with markedly undernourished children have shown decreased ability to utilize fat and starch. Presumably this decreased function is dependent on malnutrition. Since failure of fat absorption is one of the outstanding characteristics of celiac disease, it is reasonable to consider that malnutrition may be an important causative factor in the occurrence of this disease. The existence of diarrhea and digestive disturbance preceding

celiac disease in many instances offers ample opportunity for malnutrition and this particular type of functional digestive disturbance.

Symptoms

Celiac disease develops sometime after the infant has been weaned, usually during the second year after birth. The onset may be insidious. The infant ceases to gain in weight, becomes irritable, the abdomen distends, and the stools gradually assume the features characteristic of the disease. In other instances the onset is related to an attack of diarrhea from which the infant never recovers completely. When the condition is fully developed, the stools are bulky, light-colored, greasy in appearance, frothy, and extremely foul-smelling. They consist chiefly of fatty acids and soaps, with small amounts of neutral fat. Undigested food particles may be seen. Blood or pus is not present. The light color of the stools is not due to absence of bile pigment, as this may be detected chemically.

The number of stools varies from four to eight daily, but during acute exacerbations of the disease the stools may become much more numerous. Alternating with severe diarrhea are periods in which the stools have a fairly firm puttylike consistency. They are still bulky and foul-smelling, however, and always contain an excess of soaps if fat is being fed.

The appetite is variable. Almost complete anorexia may persist for days at a time and then the child may appear ravenously hungry, yet may refuse food when it is placed before him. Vomiting is not a prominent symptom. The tongue is pale and may be glazed and fissured.

The somatic symptoms are those of food deprivation produced by failure of absorption. Deficiency disease of almost any type may appear. Unless the condition is brought under control, the infant wastes to an extreme degree. Subcutaneous fat disappears throughout the body.

The wasted extremities are in striking contrast to the greatly distended abdomen, giving to the infant a peculiar spiderlike appearance.

Edema is present frequently and is dependent on a low concentration of albumin in the blood plasma, not to renal or cardiac disease. Progressive muscular weakness and wasting occur. Children who may have been walking often become bedridden. A moderate degree of secondary anemia usually is present. The calcium and phosphorus of the blood may be lower than normal.

Growth of the long bones becomes retarded so that the child at the age of five or six years may be no taller than a normal child of two years. Osteoporosis is commonly observed, but growth usually is too slow for rickets to develop. Some of the changes in the bones may be similar roentgenographically to those observed in rickets. The term "celiac rickets" has been applied to the condition.

Tetany may occur. Scurvy is sometimes seen as a result of dietary restriction. Glucose tolerance curves usually are flat, due to slow absorption from the intestinal tract. Children with celiac disease are likely to be irritable and fretful and difficult to manage. Because of failure of muscular performance expected for the age, mental backwardness may be suspected, though none occurs as a result of the disease.

The condition is a chronic one, lasting for years unless brought under control by dietary means.

Treatment

The essential factor in the treatment of celiac disease is dietary regulation. These children have intolerance for fat and for starches. This intolerance appears to be due not to lack of pancreatic enzymes, but primarily to difficulty in absorption. It is claimed that the difficulty lies in lack of phosphorylation of glucose and the fatty acids, but this theory is not yet proved. At the onset of the condition the intolerance for complex carbohydrate appears first.

As the condition develops, intolerance for fats subsequently appears. As improvement occurs under dietary management, it becomes possible first for the child to tolerate fats and only later the complex carbohydrates. In a few instances intolerance to only one of these food factors is present.

Starch-containing foods should constitute no part of the diet. The polysaccharides in a general way may be expected to aggravate the disease. Monosaccharides are well tolerated and may be fed in large amounts to satisfy the carbohydrate and energy needs. As the carbohydrates of fruits are largely monosaccharides, fruits and fruit juices can be taken freely. Orange juice may be administered with safety. Ripe bananas are especially well tolerated despite their content of sucrose, and commonly have a prominent place on the therapeutic diet because of their high content of simple carbohydrates in solid and easily digestible form, and their relative lack of irritating undigestible residue.

The child with celiac disease may take protein in large amounts with impunity and protein of necessity must form the basis of the diet. The protein of skimmed milk, egg white, meat, fish, chicken, or liver may be given. Because of the lowered gastric acidity that is so often a feature of celiac disease, acid milks are better tolerated than sweet milk, though in most instances boiled sweet skimmed milk is entirely satisfactory.

According to the plan of treatment recommended by some, protein milk is the basis of the diet and constitutes the sole diet for three or four weeks, or until the stools have become firm and the child has begun to gain weight. Satisfactory results may be obtained with the protein milk diet, especially in instances of milder disease, but protein milk contains more fat than is desirable to give in the more severe disease, and beneficial effects are obtained more promptly even in the mild disease when less fat is fed. Also a "complete" diet is desirable from the beginning.

and protein milk alone does not qualify in this respect. With properly chosen foods the diet may be made complete, a more appetizing and somewhat more liberal diet is possible, and improvement in the condition of the child may be noted almost at once. Such a diet may consist of the following foods:

Boiled skimmed milk (or acidified skimmed milk) 720 to 1000 ml. (24 to 32 ounces).

Cottage cheese from skimmed milk 30 grams (1 ounce)

Egg whites 2 or 3 (coddled—cooked slowly in water just below the boiling point)

Sieved boiled liver 30 to 50 grams (1 to 1½ ounces)

Baked ripe banana or very ripe raw banana 1 or 2

Strained orange juice 250 ml. (8 ounces)

Strained tomato juice 250 ml. (8 ounces)

Cod-liver oil 1 teaspoonful (300 to 400 units of vitamin D) or a concentrate of vitamins A and D dispersible in milk

The total amount of food is divided into four feedings for the day.

This diet is supplemented with dextrose which is given in 10 per cent solution flavored with orange juice. As much as 125 to 200 milliliters (4 to 6 ounces) of this solution may be given at hourly intervals between meals. If well taken, the amount of dextrose may be increased beyond the amounts stated; a total of 200 grams usually is taken easily. The basic diet supplies all nutritional essentials except adequate energy. By means of dextrose additions a high energy intake is possible, leading to more rapid weight gain and more rapid recovery from the disease.

With a diet such as the one discussed, fully adequate in calories, minerals, and the known vitamins, the nutrition of the child should improve rapidly, the stools should become relatively normal, and the appearance of health should be regained rather quickly. Although gain in weight may begin at once, growth in height may not be

observed for several weeks. Once growth in body length starts, it progresses rapidly until the normal height for the age is approached.

A child treated in this manner soon reaches a stage of improvement at which he seems as normal as other children, but the tolerance for fat and complex carbohydrate returns slowly. Continuous improvement occurs only if the diet is constantly low in fat and complex carbohydrate. Early in the course of management, one cracker or one spoonful of potato may cause a digestive upset of several days' duration.

After weeks of restricted diet, or even several months, when the disease is severe, fat may be added cautiously. The first change is from skimmed milk to half skimmed milk, then to whole milk. Whole egg may be given instead of egg white. Before complex sugars are added to the diet, variety and relief from monotony may be attained by additions of moderate amounts of well-cooked green vegetables (not starchy varieties) and of strained cooked ripe fruit other than banana and orange. Tolerance for starch returns late, sometimes a year or more of progressive improvement being necessary before appreciable amounts of starch may be given. The first additions should be made tentatively and most cautiously. Since complex sugars and starches are the last additions to the diet, the gradual increase in these materials leads to a normal unrestricted diet. The duration of the period of management before this stage is reached is widely variable and may be as long as two years or even more.

The role of various vitamins has been studied in an attempt to discover the cause of celiac disease. In malnutrition as general and severe as occurs in celiac disease, the addition to the diet of any one component in an easily absorbable form may be expected to result in improvement. What is needed is a dietary regimen adequate in all respects, not just a few. Too much emphasis on one phase of the regimen often leads to neglect of other and equally necessary phases.

In the treatment of celiac disease it is essential that proper attention be paid to the general hygienic surroundings of the child. Fresh air, sunlight, proper clothing and sufficient rest all tend to promote recovery. These children are difficult to manage, and remain fretful and querulous until their nutrition becomes much improved. Whoever cares for them should be even-tempered, considerate, and possessed of infinite patience. Harsh treatment may cause the child to lose the appetite for days. On the other hand, these children must not be spoiled by excessive indulgence. The diet must be continued persistently, and no variations should be made only for the purpose of satisfying the child. With proper treatment, children with celiac disease may be expected to reach completely normal mental and physical development.

Cystic Fibrosis of the Pancreas

(PANCREATIC INSUFFICIENCY, PANCREATIC FIBROSIS)

Cystic fibrosis of the pancreas is a disease in which the outstanding abnormalities are in the pancreas and in the bronchi. The pulmonary lesion is not represented in the name of the disease, but it is a prominent part of the clinical picture. When the disease of the pancreas has advanced sufficiently, the symptoms resemble closely those of celiac disease.

The underlying abnormality in this disease is inspissation of body secretions. Thickened secretion in the pancreatic ducts interferes with the flow of digestive secretions of the pancreas into the digestive tract. The parenchyma of the pancreas eventually undergoes cystic and fibrotic change. The lack in the intestinal tract of the digestive secretion of the pancreas causes indigestion and nutritive failure. Inspissated secretions in the bronchi tend to occlude some of the smaller bronchi and to lead to infection of the affected areas. At times the liver may be affected also because of obstruction of its ducts. The clinical findings vary depending on which organs are most severely affected.

In a few instances the pancreatic disease is well advanced at birth. In these cases the meconium is often too thick to be passed on and the baby dies of meconium ileus. Pancreatin added to these thick secretions changes them to a thin watery fluid that could be moved along the intestinal tract easily.

The majority of children with fibrocystic disease of the pancreas show evidence of it in early infancy. When it appears early the respiratory difficulties dominate the picture and the infant rarely survives the first year.

When the disease is less severe, the respiratory disturbance is less marked than the nutritional disease and the symptoms resemble those of celiac disease. These children do not improve with the dietary therapy for celiac disease. Digestion and absorption of all food are sharply decreased; the volume of the intestinal content is increased, causing abdominal distention and excretion of large loose stools that are foul. These children fail to absorb not only fat and complex carbohydrate, as in celiac disease, but they also have abnormal amounts of protein in the stools. Often the fecal fat is unsplit, but considerable hydrolysis of the fat may occur if intestinal lipase is present. Symptoms of vitamin A deficiency may be marked because of failure of absorption.

The pancreatic secretion that reaches the duodenum contains very small amounts of digestive enzymes; the secretion itself is thick and mucilaginous. Examination of this secretion is the most certain method of differentiating cystic disease of the pancreas from celiac disease. The placing of a duodenal tube to obtain secretion should be checked by fluoroscopy to be certain that aspirated fluid does not represent gastric content. Aropy secretion that sticks to a glass rod is characteristic of cystic disease of the pancreas, as is a secretion lacking in trypsin.

Another procedure useful in diagnosis is determination of the amino acid content of the blood before and at intervals for five hours after ingestion of a predetermined

amount of protein. In the case of normal infants the amino acid content of the blood increases after ingestion of protein and reaches a maximum in approximately two hours. Infants with cystic disease of the pancreas show little or no increase of amino acids in the blood.

Lacking facilities for examining duodenal secretion or for determining the amount of absorbed protein and fat, differentiation between cystic disease of the pancreas and celiac disease must be made on the basis of therapeutic trial of celiac dietary therapy, which is unsuccessful in cystic disease of the pancreas and uniformly successful in celiac disease.

One method of management of the nutritional phase of cystic disease of the pancreas is to give the same type of diet as is useful in celiac disease, and in addition to give enteric coated granules of pancreatin. Another procedure is to give a food that requires no digestion by pancreatic enzymes. Such a food would be amino acid solutions fortified with dextrose, minerals and vitamins. Correct fortification of such a food to make it a complete diet is not simple. With effort it may be possible to maintain the nutrition of these children. In such case the prognosis would then depend much on the status and progress of the respiratory disease.

Cystic disease of the pancreas should always be considered as a possible diagnosis in the presence of malnutrition accompanied by diarrhea or increased volume of stool and in chronic infections of the respiratory tract that are refractory to treatment. The disease is not rare. The incidence of this diagnosis at autopsy is reported to be from one to five per cent. Though the disease is predominantly one of infancy and early childhood, it may come to attention at a later time. The oldest child found recorded was fourteen and one-half years of age.

CHAPTER XXIII

VOMITING

The most frequent causes of vomiting in infancy are:

1. Overdistention of the stomach by swallowed air.
2. Too frequent feeding.
3. Too large volumes of food.
4. Unsuitable composition of the food.
5. Improper clothing and handling.
6. Parenteral infections.
7. Habit or "nervous" vomiting; rumination.
8. Gastroenterospasm.
9. Obstruction of the gastrointestinal tract
 - Atresia of the esophagus
 - Pyloric stenosis
 - Obstruction of the duodenum
 - Intussusception
10. Anhydremia.
11. Allergy.
12. Intracranial conditions.
13. Toxic states.

Vomiting Due to Swallowing of Air

All young infants swallow a certain amount of air during and between feedings. Roentgenograms almost invariably reveal an air bubble in the stomach. Infants who are underfed and consequently always hungry are especially likely to swallow large amounts of air. More air is swallowed when an infant takes a feeding from a bottle while lying on the back than when he nurses the breast or is fed in a semiupright position. When an infant having a large bubble of air in the stomach is fed, he is likely to take a volume of food which, together with the air already present, is in excess of the gastric capacity so that

something must escape. If the infant is lying on the back, the air will accumulate anteriorly in the stomach above the level of the cardiac orifice, so that milk will be expelled until the distention of the stomach is relieved. If, on the other hand, the infant is held in an upright position, the air bubble will rise to the cardiac end of the stomach, and be belched up. In order to prevent the vomiting which occurs as the result of swallowed air, the infant should be held over the shoulder just before and just after each feeding and patted on the back until belching occurs. It may be necessary to interrupt the feeding in order to get rid of the air. In the case of some infants who swallow large amounts of air between feedings, it is necessary to keep the infant constantly propped up in bed in a semiupright position. This is easily accomplished by resting the infant's back against pillows and holding him in position by means of a small harness made from tapes fastened to his body and to the sides of the crib. This does not usually interfere with the infant's sleep.

Some infants develop the habit of finger sucking, and in this way swallow a great amount of air. Finger-sucking may be discouraged or prevented by encasing the elbows in lightweight splints, by pinning the sleeves to the bed clothes or diaper, or by covering the hands with aluminum ball mitts or celluloid cuffs. An excellent procedure is to place a favorite toy in the hand the fingers of which are being sucked.

Overdistention of the Stomach by Too Frequent or Too Large Feedings

Considerable variation is noted in the emptying times of infants' stomachs and is dependent on constitutional factors as well as on the character of the food. When feedings are given at such short intervals that one feeding has not passed out of the stomach before the next is given, overdistention is likely to occur, with consequent spitting

up or vomiting. Vomiting is much more frequent in the case of infants fed at two- or three-hour intervals than in those fed at four-hour intervals.

The giving of too large volumes at a feeding results in overdistention of the stomach, especially when the feedings are taken very rapidly. Vomiting from this cause is seen especially in artificially fed infants receiving very dilute milk formulas, for in such instances the intake of large volumes is necessary in order to meet the nutritional demands and to satisfy the infant's hunger. Vomiting from this cause may be remedied by the administration of a more concentrated food. A frequent mistake in infant feeding is to increase the dilution of the food in the case of infants who are vomiting when they are already taking too large volumes of a food which is too dilute. In some instances infants have a gastric capacity smaller than usual. In such cases the feeding of amounts customary for the age causes distention and vomiting. A more concentrated food is indicated.

Vomiting Due to Unsuitable Composition of the Food

Feedings containing a large proportion of fat leave the stomach slowly, so that complete emptying may not occur before the next feeding is given. Such conditions predispose to vomiting. Vomiting from this cause usually is not seen in well infants except when top milk or cream mixtures are fed, as the amounts of fat in whole milk are not sufficient to delay greatly the emptying of the stomach. When infants are ill from any cause, gastric motility is decreased and food tends to remain in the stomach abnormally long. Under these conditions the fat of the food, when present even in customary amounts, is likely to be broken down with the formation of irritating products, such as butyric acid, and vomiting is more likely to occur than if no fat were present.

Some infants with more than the average amount of gastric acid secretion vomit when given acid milk but are

able to retain sweet milk. The feeding of raw milk, or milk which has been heated to only a moderate degree, results in the formation of very large curds in the stomach, which pass the pylorus with difficulty. Excessive size of curds is one of the causes of vomiting, easily remedied by using boiled, evaporated, dried, or acid milk. Spoiled food or unusual articles of diet may of course cause vomiting.

Vomiting Due to Improper Clothing and Handling

The infant who is overclothed, especially when tight abdominal binders are applied, is likely to vomit. The infant should be allowed to rest after feeding. If he is picked up, played with, and trundled around, he may vomit.

Vomiting Due to Parenteral Infections

In the presence of any acute infection, vomiting may occur. Infants suffering from otitis media and pyelitis are especially likely to vomit. In beginning otitis media, vomiting may be noted before any rise of temperature has occurred and before any local evidences of infection in the ear are detected. Infants with chronic pyelitis may vomit at short intervals for months at a time. Almost any other infection in the body may at times lead to vomiting.

If an infant has been taking reasonable amounts of a well-balanced formula, at proper intervals, and then suddenly begins to vomit, the first step should be to look for evidences of infection rather than to assume that the feeding is at fault. Certain measures should be taken, however, to control the vomiting while the infection is being treated. The fat content of the formula should be decreased. If the vomiting is associated with diarrhea, the amount of sugar also should be decreased or dextrose should be used as the added sugar. Thus, boiled skimmed milk may be the basis of the diet. Acidification of the milk is useful for several reasons, especially for the young baby.

Habit or "Nervous" Vomiting; Rumination

Some infants vomit easily on the slightest provocation. This is especially likely to occur in infants of the nervous, fretful type, and in those who are continually handled and played with. Other infants develop the habit of bringing up food voluntarily. The food merely may be brought up into the mouth, held for a while and swallowed, or may be expelled completely. This habit is known as "rumination." There appears to be no organic basis for the condition. Some infants appear to ruminate just to amuse themselves; others, to attract attention. The infant usually goes through a series of grimaces and contortions just preceding the vomiting; he frowns, smiles, works his jaws backward and forward, stiffens his body, arches his neck and expels the food. Occasionally vomiting is initiated by putting the hand in the mouth. Rumination is a habit which may continue throughout infancy and well into childhood and one which may result in a severe degree of undernutrition. It is important that rumination should not be confused with other forms of vomiting.

There are various means of treating rumination, and no one means is effective for every infant, and with some all methods of treatment may fail. One of the most effective means of treatment consists in thickening all feedings given by boiling with cereals. The milk formula may be the same as in the case of a normal infant except that approximately 8 per cent of barley or other cereal flour is added and the whole is cooked for fifteen minutes in a double boiler. Such a mixture should be so thick that it will not fall from an inverted spoon. Any vegetables or other articles of diet are incorporated in the thick feeding. These thick mixtures of course cannot be fed from an ordinary nipple or from a bottle, but must be given with a spoon or, in the case of young infants, from a nipple of the type used on wide-mouthed feeding bottles (Hygeia nipples). The tip of the nipple is cut off so as to leave a hole about the

diameter of a lead pencil. The body of the nipple is filled with the thickened mixture, and as the baby sucks, the food is forced down into the tip with a glass or wooden rod. These thick mixtures are vomited with difficulty, and some infants, finding themselves unable to vomit the food, after a period of time give up the attempt. The use of thick mixtures, however, may have to be continued for several months. The infant may be so accomplished in the art of vomiting that even these thick mixtures are promptly returned. It then becomes necessary to use other means of treatment.

The mouth may be kept tightly closed after feeding by the use of a chin strap (a small square of muslin attached by four tapes to a larger square covering the top of the head to which the tapes are tied tightly). There is a certain danger in the use of this contrivance because if the infant should vomit, he may aspirate a portion of the food.

In the case of infants who initiate the vomiting by placing the hands in the mouth, the arms may be restrained by loose splints, or the hands may be encased in ball-shaped aluminum mitts, or celluloid cuffs. Placing the infant on the stomach after feeding is sometimes effective, but unless retained in this position by mechanical means, older infants are likely to roll over on their backs when they are ready to ruminate.

Gastroenterospasm

Vomiting may be one of the symptoms of general gastroenterospasm. This particular phase of the subject is discussed in the next chapter.

Obstruction of the Gastrointestinal Tract

Any condition that produces obstruction of the gastrointestinal tract results in vomiting. Obstruction may be organic or functional. Functional obstructions are chiefly those caused by acute infections in the abdomen, such as peritonitis, and are the result of paralysis of the bowel

produced by the infection. Paralytic obstruction may occur also from severe toxicity from infections outside the abdomen, as, for example, pneumonia. In these instances the treatment of the obstruction is chiefly that of the causative infection.

The organic obstructions of infancy consist chiefly of atresia or stenosis produced by congenital malformations, of obstruction by peritoneal bands or persistent Meckel's diverticulum, which also are congenital malformations, and of intussusception of one part of the bowel into another. Obstruction may occur in almost any part of the gastrointestinal tract. The symptoms vary according to the degree or completeness of obstruction and according to its location. Only a few typical examples will be discussed.

ATRESIA OF THE ESOPHAGUS

The common site of atresia of the esophagus is at the level of the bifurcation of the trachea. In the common variety of this condition the two patent segments of the esophagus are connected by a fibrous cord, and a fistula exists between the lower segment and the trachea. As soon as any food or fluid is taken after birth it is regurgitated. The history of prompt regurgitation of everything taken suggests the nature of the difficulty. The diagnosis is confirmed by x-ray examination after placing iodized oil in the esophagus. By this means it is made evident that the upper segment ends in a blind pouch. The finding of air in the stomach by either percussion or x-ray examination gives evidence of the tracheal-esophageal fistula. The condition can be corrected only by surgical means. In many instances the ends of the two patent portions of the esophagus can be anastomosed and at the same time the fistula obliterated. The operation is severe and the mortality is high, but this type of operation offers better chance of success than the older procedure of trying to feed the baby by way of gastrostomy without closure of the tracheal-esophageal fistula.

PYLORIC STENOSIS

Pyloric stenosis may be of all degrees. There may be merely a tendency to spasm of the pyloric sphincter or definite hypertrophy of the circular muscle fibers of the pylorus and fibrosis with constant and practically complete occlusion of the pyloric orifice. In some cases the pylorospasm is merely part of a general gastroenterospasm.

Although at times referred to as congenital hypertrophic stenosis, the symptoms usually are not marked until after the second or third week of life; however, there may be some "spitting up" from the time of birth. Persistent vomiting from the time of birth is more likely to be due to other causes, such as duodenal atresia or stenosis. Infants with pyloric stenosis vomit even though the feedings are of good composition and given at proper intervals. Pyloric stenosis is as frequent in breast-fed babies as in those artificially fed. It is more common in males.

The first symptom noted is vomiting, which does not differ in character from that due to any other cause. Vomiting usually occurs shortly after feedings or the taking of water. The vomiting may at first consist merely of spitting up a small amount after feedings. Very soon, however, the vomiting becomes more forcible: the hypertrophied and dilated stomach becomes capable of expelling food in a projectile manner, often to a distance of several feet. The vomitus may be forced through the nose as well as the mouth. Even water is vomited.

As the result of obstruction at the pylorus, the gastric musculature hypertrophies. The normal gastric peristalsis becomes greatly accentuated until the peristaltic waves may become clearly visible. These waves always pass from the left to right and should not be confused with peristaltic waves in the transverse colon, which pass in the opposite direction. In those cases in which the stomach has become much dilated and ptosed, the waves may be seen passing downward and to the right and may cease in the neighbor-

hood of the umbilicus or below it. The waves may be present constantly, or may be seen only after the infant has taken food or water. They may at times be stimulated by applying cold to the abdomen and tapping the skin of the abdominal wall. Peristaltic waves of the stomach are observed in almost all cases of pyloric stenosis of any considerable duration; they are, however, not absolutely pathognomonic of this condition, inasmuch as some gastric peristalsis may be seen also in infants who have been vomiting excessively from other causes.

In most cases the thickened pylorus may be felt. The size of the tumor varies from that of the tip of the little finger to that of a large olive. The position of the pylorus may vary so that the tumor is not always felt in the same place. It is more frequently found slightly below the edge of the liver in the nipple line, but may be considerably lower, in the neighborhood of the umbilicus. Some idea as to the location of the pylorus may be obtained by noting the point at which the peristaltic waves disappear.

When much of the food intake is lost by vomiting, progressive malnutrition occurs, until finally the infant becomes markedly athreptic. On the other hand, even though a portion of each feeding may be vomited, enough food and water may pass the pylorus so that the nutrition is not impaired. Infants with pyloric stenosis usually are constipated, but may suffer from a starvation type of diarrhea. When the pylorospasm is a part of general gastroenterospasm, diarrhea is common.

The persistent vomiting of pyloric stenosis results in a great loss of chlorides from the body in the form of hydrochloric acid and, to a lesser extent, of sodium chloride. Because of excessive loss of chloride through the vomitus, blood chloride is lowered. To compensate osmotically for the chloride loss, excessive amounts of sodium bicarbonate are retained in the blood and a severe degree of alkalosis is likely to result. It is characteristic of alkalosis that the

breathing becomes shallow, depressed, and irregular, with frequent long apneic pauses. Symptoms of tetany may develop, consisting of general hypertonicity, carpopedal spasm, a positive Chvostek sign, or generalized convulsions. Examination of the blood shows a high bicarbonate and low chloride content. The CO_2 -combining power sometimes may be over 100 volumes per cent (47.5 milliequivalents to the liter). The calcium and phosphorus values of the blood usually are normal.

In cases of severe pyloric stenosis in which a large portion of the food is vomited, little absorption of water takes place. Consequently the urine is scanty and highly colored, and the infant becomes dehydrated. The urine contains practically no chloride. Because of retention of bicarbonate by the body, the urine is acid in reaction, even though the infant may have a severe degree of alkalosis.

Otitis media is often associated with pyloric stenosis and is probably the result of passage of forcibly vomited material up into the Eustachian tube.

In pyloric stenosis there is a tendency to ultimate recovery, provided the infant's nutrition can be maintained. The usual course of events, as judged from clinical symptoms and roentgenographic evidence, appears to be a progressive narrowing of the pyloric opening during the first month or two after the onset of symptoms, followed by a stationary period in which little change occurs. After the third or fourth month the tendency is for the pyloric aperture to become larger so that food passes more easily. The tumor, it is true, may become larger during this time, but with the growth of the stomach the pyloric opening also becomes larger, so that finally food may pass through easily.

The diagnosis of pyloric stenosis is made on the basis of the symptoms and signs already described. Fluoroscopic examination after a barium meal is an aid in the diagnosis. The infant should be examined immediately after the barium is given, and at 15- to 30-minute intervals thereafter

for the next three hours. Under normal conditions the stomach begins to empty while the meal is being taken, and should be almost completely emptied by the end of three hours. The fluoroscopic examination is of especial aid in differentiating between pyloric stenosis and duodenal stenosis or atresia, a condition giving rise to very similar symptoms.

The character of the vomitus may give information of value in the differential diagnosis of pyloric stenosis. In pyloric stenosis the amount of food vomited is large, and when the stomach has become dilated may be considerably greater than the amount of food taken at a single feeding, the residues of previous feedings being included. The vomitus is not bile-stained because the constriction of the pylorus prevents the regurgitation of bile into the stomach, whereas in other forms of vomiting, especially those due to obstruction lower in the intestinal tract, bile is often present in the vomited material.

Congenital duodenal stenosis or atresia may give rise to symptoms which are very similar to those of pyloric stenosis, but with no palpable pyloric tumor. The vomitus is usually bile-stained, and fluoroscopy after a barium meal reveals passage through the pylorus with blocking at some point in the duodenum.

The treatment of pyloric stenosis may be divided into medical and surgical methods. The choice of the method to be used will depend on the age and nutritional condition of the infant, whether or not he is breast-fed, and the degree of obstruction present. The choice of treatment will also be influenced by such extraneous factors as financial conditions, and the availability of suitable hospital facilities and surgical and nursing care.

In any case of suspected pyloric stenosis, medical and dietetic means of treatment should be tried first, but should not be continued unless some gain in weight is attained within a reasonable time. It is dangerous to allow an infant to remain at stationary weight or to lose weight while

waiting for natural processes of recovery, because once the nutrition is impaired, complicating infections are likely to occur, and the infant may finally reach such a condition that neither medical nor surgical means are effective. Very young infants, especially those who are still breast-fed and who are not retaining sufficient food to gain weight, should not be weaned in order to apply any method of treatment, but preferably should be operated on promptly. Infants who have been treated with more or less success for three or four months, whether breast-fed or artificially fed, usually do not require operation, as spontaneous improvement of the condition is the rule after this age. The application of medical means of treatment may necessitate hospital care and, at times, special nursing for long periods of time. Surgical operation requires only a short period of hospitalization and when skillfully performed is accompanied by negligible mortality in the case of infants in reasonably good nutritional condition.

The chief medical methods of treatment consist in the administration of atropine, or phenobarbital, the feeding of thickened formulas, refeeding and gastric lavage.

Atropine administration is successful in a fair proportion of cases; in others, it diminishes the vomiting somewhat but not to a degree sufficient to allow for retention of enough food for adequate nutrition. In still other cases, atropine appears to be entirely ineffective. It has been assumed that in this latter group a hypertrophied inelastic pyloric musculature is present. It is not usually possible, in an individual infant, to predict whether or not atropine will be successful; it may act well even when palpation reveals a definitely enlarged pylorus. Atropine is usually administered by mouth a few minutes before each feeding in the form of 1:1,000 dilution of atropine sulfate ($\frac{1}{4}$ grain to $\frac{1}{2}$ ounce of water). Since the potency of solutions as ordinarily prepared by the druggist may vary considerably, the dose of each new solution should be determined by the reaction of the infant. An initial dose of 1:1,000

solution is 0.06 ml. (1 minim), equivalent to 0.06 mg. (1/1,000 grain). The dose is then increased 0.06 ml. (a minim) at a time, until the physiologic effect is observed, which consists of a diffuse blush of the skin occurring within ten or fifteen minutes after the administration. A dosage very slightly less than this amount should be continued and given before each feeding. The average amount of atropine solution required to produce the physiologic effect in an infant one month of age is 0.12 to 0.18 ml. (two to three minims), but infants vary greatly in their susceptibility to atropine. Some infants flush with 0.06 ml. (one minim) and others require as much as 0.30 or 0.35 ml. (five or six minims); this latter dosage should not be exceeded. Often the infants seems very drowsy for some hours after the first few doses, but this should not prevent further administration. The atropine solution is measured with a minim or other standard dropper, added to a teaspoonful of water, and given through an empty nipple fifteen or twenty minutes preceding each nursing or bottle feeding. It may be necessary to continue the administration of atropine for several months. The administration of atropine at times leads to an increase in body temperature, which may be as high as 39° or 40° C. (103° or 104° F.) and which cannot be accounted for by the presence of infection. It is important to bear this fact in mind. The occurrence of "atropine fever" is not necessarily a contraindication to the use of atropine, although it is usually well to diminish the dosage somewhat.

Phenobarbital has the effect of allaying vomiting. In beginning treatment with phenobarbital, a dosage of 16 mg. (1/4 grain) is given before each feeding. After the infant is well under the influence, the dosage is reduced to 8 mg. (1/8 grain). The dosage should be slightly less than that which makes the infant definitely drowsy.

Another therapeutic measure consists in the administration of food thickened by boiling with cereal. (See p. 337.) Such thick mixtures are not as easily vomited as are cus-

tomary feedings. The stomach by its peristaltic action appears capable of forcing these thick mixtures through the pylorus.

It is usually advisable to apply the method of thick cereal feeding only in the case of babies who already have been weaned. A baby should not be weaned in order to feed him thick cereal, although the breast milk may be expressed and thickened with cereal. It is difficult, however, in such circumstances to maintain the milk secretion, and ultimately weaning is likely to become necessary. In the case of the bottle-fed baby, the formula may be of the same type as that given to normal infants of the same age, except that it is thickened. In general, a concentrated formula, that is, one having a high caloric value to the unit volume, is indicated in order that the total volume of the food given may be small.

Some infants do better when fed at long intervals (three to four hours), but occasionally excellent results are obtained when amounts of only an ounce or so are given at hourly intervals. The feeding interval selected should be that which permits the greatest total amount of food to be retained in a day. Although most babies retain thick formulas better than the ordinary liquid types of feeding, an occasional infant vomits more when receiving the thickened feedings.

In using concentrated formulas thickened with starch, there is some danger that the infant will receive an inadequate amount of fluid. Water should not be given by mouth very soon after the feedings, but if small quantities of water are given at intervals they may be retained. It is usually necessary, however, when concentrated thickened feedings are given, to administer fluids parenterally. Physiological sodium chloride solution not only supplies water, but also the chloride necessary to replenish the depleted blood chloride.

In the case of infants who are being fed at the longer intervals, refeeding immediately after vomiting has oc-

curred is a valuable means of introducing sufficient food to provide for the infant's nutrition. Refeeding should be attempted only when the vomiting occurs within one hour after the time of feeding. The volume of food vomited is roughly estimated, and this amount of the same formula is given immediately after the vomiting. In such cases the feeding is often retained. In this way, even though the infant vomits after each feeding, it may be possible to introduce and have retained in the course of a day a sufficient amount of food. In breast-fed infants the refeeding may consist of a formula, or if the mother has an abundant supply of milk, expressed breast milk. In general, however, refeeding is not satisfactory in the case of breast-fed infants.

Gastric lavage with a 1 per cent solution of sodium bicarbonate just previous to feeding serves to remove accumulated mucus and to allay gastric irritability. After the lavage, the feeding may be given through the same tube. In some infants, tube feeding appears to stimulate peristalsis less than feeding by mouth.

A combination of the methods mentioned above may be used. The infant may be given thickened feedings and atropine before each feeding. Refeeding may be resorted to when vomiting occurs. Phenobarbital, together with atropine, may be given when concentrated liquid feedings are used.

The nonsurgical methods of treatment outlined above are more likely to be successful when they are instituted early than when they are undertaken after the infant's nutrition has suffered and the stomach has become dilated and hypertrophied. Medical means of treatment suffice in a good many cases in which there is no definite hypertrophy of the pylorus, the condition being essentially pylorospasm. It is claimed by some that in a very high proportion of cases pylorospasm and pyloric stenosis may be successfully treated without recourse to surgery. It should be noted, however, that many babies so treated have to be

kept in hospital wards for a long period of time, that the nutrition may be severely impaired, and that secondary infections, especially otitis media, are of frequent occurrence. It is not generally advisable to persist with medical means of treatment unless after a short trial the infant shows definite signs of improvement and is beginning to gain at a reasonable rate. Medical treatment should not be continued until the infant becomes badly undernourished and a poor surgical risk. In our own experience, the mortality from pyloric stenosis is much lower when all those severely affected are treated promptly by surgical means.

A number of surgical procedures have been used for the treatment of pyloric stenosis. The most satisfactory is the simple pyloroplasty originally used by Fredet and later developed by Rammstedt. In the case of infants in good nutritional condition, the operative mortality is practically nil, and prompt disappearance of symptoms follows. The results of operation, however, are disappointing unless proper attention is given to the preoperative and postoperative care of the infant. In the case of infants who have been badly fed or in whom the condition has lasted for a long time until the nutrition is poor, the stomach is greatly distended, and the water balance is disturbed, operation is not so uniformly successful.

Operation should not be attempted while the infant is badly dehydrated and suffering from alkalosis. Fluids should be replenished through the parenteral administration of salt solution. Intraperitoneal injections, however, should not be given within twenty-four hours preceding operation. If the infant is in poor nutritional condition, intravenous injection of dextrose is indicated, and transfusions are of value. No feeding should be given within four hours preceding the time of operation.

The operation is preferably performed under local anesthesia. Customs in postoperative management differ, in that some physicians offer food within a few hours of the operation, while others do not give a milk feeding until at

least twenty-four hours have elapsed. When early feeding is employed, vomiting is somewhat more likely to occur. One successful regimen is as follows. Nothing is given by mouth for approximately eighteen hours; during this period fluids may be given parenterally. Then dextrose in 5 per cent solution is given by mouth, 15 ml. (one-half ounce) every hour for two or three feedings, followed by 25 to 30 ml. (one ounce) every hour for two or three feedings. If the baby had been breast-fed previously, he may then be put to the breast and the regular feeding schedule may be resumed fairly rapidly. During the period when the baby is not receiving food, the mother's milk supply should be maintained by expression of the milk. In the case of the artificially fed baby, the formula should be somewhat weaker at first, or of slightly less volume than he would be getting normally. An acidified formula may be of definite advantage. After a period of gradual increase in the food for three or four days, the baby usually can take without difficulty a formula customary for the age.

Obstruction of the Duodenum

Obstruction of the duodenum occurs less frequently than does pyloric stenosis, the condition with which it is most likely to be confused. Obstruction may be from atresia, stenosis, or pressure from a peritoneal band, all being congenital malformations. The symptoms of complete obstruction are the same regardless of cause. When obstruction is partial, as with stenosis, the severity of the symptoms depends on the degree of obstruction, but in any case surgical relief is necessary.

Obstruction is most likely to occur in the third part of the duodenum. The symptoms of duodenal atresia are present immediately after birth, the vomiting being of the same character as that observed with pyloric stenosis. Distention and hypertrophy of the stomach occur. Gastric peristaltic waves are seen, but no pyloric tumor is palpable. The vomitus is bile stained, a feature distinguish-

ing the condition from pyloric stenosis. Such stools as there may be retain their meconium character. The various symptoms make it obvious that an obstruction is present, although its location may not be evident until operation. X-ray examination may assist in locating it.

When the obstruction is partial, the symptoms are the same as those of complete obstruction, but of lesser severity. Vomiting is an outstanding feature. Gastric peristalsis may be present. The stools show evidence of some food having passed the obstruction. X-ray examination, using a barium meal, often is misleading in that no obstruction is shown. After an interval, barium is present on both sides of the obstruction and the site of the obstruction is not evident. The diagnosis must be made on the basis of the history and physical examination.

Intussusception

The most common variety of intestinal obstruction in infancy is intussusception. It is a disease chiefly of infancy. More than one-half of the cases occur in the first year and three-fourths of them under two years. Males are affected in approximately two-thirds of the cases and in most instances the child is one who had been robust and healthy.

Intussusception is a condition in which one part of the intestine becomes invaginated in another part distal to it. In approximately 85 per cent of the cases the ileum is invaginated into the cecum. The invaginated intestine carries with it its mesentery and blood vessels. With the cutting off of the blood supply the bowel becomes strangulated with resulting edema, then hemorrhagic changes and finally gangrene. Eventually peritonitis develops if the infant lives long enough.

The onset is sudden with severe pain. The pain recurs as paroxysmal colic when new portions of the intestine become invaginated and the mass increases in length. In

other varieties of complete intestinal obstruction constipation is absolute. In intussusception fecal matter is pushed ahead of the advancing invaginated bowel. First one or two loose fecal stools are passed, then blood or blood and mucus. Blood may be expected to appear within about six hours of the onset.

Vomiting is an early symptom. At first the vomitus consists of gastric content; subsequently it contains bile; seldom is it fecal in infancy. Soon after the onset the infant becomes restless. Soon prostration appears and eventually collapse with pallor. After the second or third day the temperature begins to rise. For the first day or two the abdomen is relaxed and the sausage-shaped tumor mass of the affected bowel can be felt. Subsequently distention occurs.

The diagnosis is made by the character of the onset, palpation of the affected bowel, and passage of blood from the anus. Rectal examination reveals nothing other than blood and mucus on the examining finger, except that in a few instances enough bowel has become affected that the distal end of the tumor may have come within reach of the examining finger. Few conditions are likely to be confused with intussusception. Dysentery may produce some resemblance because of bloody stools. In case of doubt as to diagnosis fluoroscopic examination while giving a barium enema is likely to prove helpful.

The only treatment to be recommended is surgical. Intussusceptions have been known to reduce spontaneously and some success has been reported with the use of enemas under pressure. Relief by means other than surgical is not to be relied on. What is done at operation depends on the condition of the affected bowel. If the operation is done early, the intussusception can be reduced. Later when the bowel has become gangrenous, it must be resected. The mortality is low with early operation. Intussusception does not recur.

Anhydremia

Vomiting is a frequent symptom in cases of severe desiccation of the body, or anhydremia, however brought about. Dehydration is induced in prematurely born babies more easily and from more minor causes than in babies born at term and the results are more serious. Even moderate dehydration of the prematurely born baby produces vomiting with risk of aspiration. The treatment of vomiting caused by dehydration consists of restoration of normal fluid balance. (See p. 299 and p. 484.)

Allergy

Vomiting may be one of the symptoms of food allergy, and may call for the elimination from the diet of the offending proteins. (See p. 371.)

Intracranial Conditions

In infants, as well as in older persons, vomiting is an accompaniment of intracranial abnormalities, such as meningitis or brain tumor. Because the sutures are not ossified, however, they are easily separated, and consequently the intracranial pressure is never so great as in older persons, and projectile vomiting is less frequent and less characteristic.

Toxic States

In any severe toxemia, whether due to infection or to such metabolic disturbances as are seen in the presence of uremia or diabetic coma, vomiting may occur.

Acidosis is usually considered as an important cause of vomiting. As a matter of fact, acidosis in itself rarely causes vomiting. In cases of cyclic vomiting (which are usually seen after the age of infancy), vomiting is to be attributed to a general metabolic disorder and not to the acetone body acidosis which is occasionally present, for infants with a much more severe degree of acidosis than that usually observed accompanying cyclic vomiting may

not vomit at all. The faulty assumption that vomiting is a frequent result of acidosis has led to the indiscriminate administration of sodium bicarbonate to many infants showing the symptom of vomiting. This type of therapy is capable of producing definite harm, inasmuch as vomiting is likely to be complicated by alkalosis because of the loss of acid in the vomited material. The administration of alkali serves only to aggravate the condition and may even be an important contributing factor in bringing about a fatal termination.

The Effects of Vomiting on the Body

Vomiting, if severe or prolonged, may result in serious damage to the body as a result of starvation and alterations in chemical equilibrium. The chief effects of vomiting are:

1. Partial starvation, through loss of ingested food.
2. Loss of water.
3. Loss of mineral salts, especially chlorides.

So much of the food taken may be lost as the result of vomiting that partial or complete starvation results, and consequently impairment of nutrition. Very little water is absorbed from the stomach, and furthermore considerable amounts of fluid are secreted into the stomach as gastric juice. Ingested fluid and that secreted may be lost and lead to depletion of the water reserves of the body, and thus may bring about the development of desiccation or anhydremia, as in water deficit from any other cause. The subject of anhydremia is discussed elsewhere (p. 281).

The gastric juice contains hydrochloric acid and sodium chloride. When gastric secretions are lost in the vomitus, the chlorides of the body become depleted. The loss from the body of the chloride ion is almost invariably compensated for in large part by retention of the bicarbonate ion in the blood and tissue fluids, leading to alkalosis of varying severity. Such a shift toward the alkaline side is just as serious as a commensurate shift to the acid side, such as occurs in acidosis.

The characteristic findings in the blood are: a shift in reaction toward the alkaline side, a marked depletion in the blood chloride content, and an increase in the blood bicarbonate, the volume per cent of carbon dioxide, as determined by the Van Slyke method, sometimes rising to 100 (45 milliequivalents to the liter) or more. Although the blood is more alkaline than normal and has a higher bicarbonate content, the urine may be strongly acid, due to the fact that bicarbonate is not excreted in the urine but is retained in the body. The urine is also characteristically deficient in chlorides. After the therapeutic administration of chlorides, the chloride content of the blood rises, bicarbonate is excreted in the urine, rendering it alkaline, and the bicarbonate of the blood falls to normal levels and the reaction shifts back to the normal point.

When alkalosis occurs, certain characteristic symptoms may develop. These are depressed, irregular respirations, and tetany. At times there may be long apneic pauses associated with the development of cyanosis. The symptoms of tetany (see p. 440), carpopedal spasm, Chvostek sign, and generalized convulsions, may all be present.

It is thus seen that the changes in the body which result from loss of fluids by way of the vomitus are quite different from the changes which result from loss of fluids by way of the bowel, as in diarrhea. In both instances anhydremia occurs, but in one there is depletion of fixed base with the development of acidosis, and in the other, depletion of chlorides with the development of alkalosis.

When both vomiting and diarrhea occur and there is loss of both chlorides and base, either acidosis or alkalosis may result, depending on the magnitude of the loss by the different routes. When the vomited material consists almost entirely of gastric contents, as a result of high intestinal obstruction or pyloric stenosis, the more severe grades of alkalosis are observed. When the vomiting is due to obstruction lower in the intestinal tract, so that gastric juice, bile and pancreatic secretions are lost in the vomitus, the

alkalosis will be of a less marked degree; or there may be no alkalosis, the loss of base balancing the loss of acid.

When vomiting has been so severe that the body is dehydrated, prompt restoration of lost fluids is essential; in addition, chlorides should be administered in order to restore the depleted chloride content of the blood and tissues. Ringer's solution or normal saline should be given intravenously, subcutaneously, or intraperitoneally, in large amounts. This supplies not only fluids but the needed chlorides.

When, as the result of severe alkalosis, symptoms of tetany are present, these may be relieved by the breathing of a mixture of 30 per cent carbon dioxide in oxygen, or by the injection of 5 per cent calcium chloride intravenously in an amount equivalent to 3.0 ml. for each kilogram of body weight. This amount supplies not only calcium but also chloride and will reduce the carbon dioxide combining power by approximately 10 volumes per cent.

In a few instances alkalosis is so extreme that correction by administration of saline requires many hours. In such instances more rapid correction becomes desirable, even in the absence of tetany. In these circumstances ammonium chloride can be given intravenously. An appropriate amount is 3 ml. of a 5 per cent solution for each kilogram of body weight for each 10 volumes per cent of carbon dioxide combining power reduction desired.

CHAPTER XXIV

COLIC, FLATULENCE, AND GASTROENTEROSPASM

In its widest application, the term colic is used to describe any form of paroxysmal abdominal pain. Colic in infancy is practically always gastrointestinal and is caused by overdistention, forcible peristaltic contractions, or a combination of the two factors.

Overdistention of the intestinal tract may be the result of swallowing of air or excessive fermentation of undigested food, especially carbohydrates. Painful peristaltic contractions may be the result of indigestion, intestinal distention, irritation, hunger, exposure to cold, food allergy, or an instability of the autonomic nervous system.

Intestinal distention is due more frequently to swallowed air than to the gases resulting from fermentation, since the latter, consisting largely of carbon dioxide, are absorbed much more quickly than is air. Air swallowing often is the result of underfeeding. The infant who is underfed is always hungry and consequently sucks on everything he can get into his mouth. In this way he swallows much air which distends the stomach and passes through the pylorus into the intestines. Hunger, furthermore, results in a general hypertonicity of the gastrointestinal tract. Forcible hunger contractions of the stomach occur and are transmitted to the intestines. These "hunger pains" are a frequent cause of colic. Various forms of irritation may set up painful peristaltic contractions. The taking of cold food and exposure of the body to cold are frequent factors. Irritating products may be produced in the intestinal tract when diets containing excessive amounts of fat or easily fermentable carbohydrates are fed.

Certain infants suffer chronically from flatulence and colic even when given good diets. These infants appear

to have a constitutional imbalance of the autonomic or vegetative nervous system. The condition has been described in the French and German literature under the name of "neuropathic diathesis." Haas has adopted the term "hypertonic infant" to describe these babies. White has designated the same condition by the symptomatic term "gastroenterospasm." Infants of this type are likely to be seen in families who have a distinct neurotic taint. The infants are nervous, wakeful, and tense; they become pale or flushed on the slightest provocation. Spitting up of food is common, and definite waves of gastric and intestinal peristalsis are frequently seen. The abdomen is always more or less distended. There may be constipation due to spasticity of the anal sphincters, or diarrhea due to increased peristalsis. Fluoroscopic examination may show the intestinal contents hurrying rapidly downward to the lower bowel where stasis occurs. Infants with gastroenterospasm are always "colicky" and "gassy." The colic may be accentuated by underfeeding or by the presence of infection. Food allergy may lead to all the symptoms of gastroenterospasm.

Colic is more frequently seen during the first three or four months than later. One of the chief reasons for the frequency of colic during the early months is that the food requirement approaches more closely the tolerance for food than later and indigestion is more easily induced. The feeding of a formula relatively rich in fat or carbohydrate may result in indigestion with irritation of the intestinal tract. Also underfeeding may cause hunger colic. In breast-fed infants the secretion of the mother's milk may not increase as rapidly as does the need of the infant.

In the presence of pain anywhere in the body, infants are likely to show symptoms referable to the abdomen; for example, an infant with acute otitis media may show every evidence of abdominal pain.

The infant with colic screams incessantly. The face becomes suffused, the arms and legs are drawn up and often tremble. The abdomen is distended and rigid during the paroxysms but relaxes in the intermissions. In cases of simple colic there are no evidences of infection, such as fever or leukocytosis, and no localized findings in the abdomen.

Treatment

In the presence of an acute attack of colic symptomatic treatment is indicated, but this should never replace treatment directed to the removal of the underlying cause.

During an attack of colic the baby's position should be changed so as to allow accumulated air to escape. The infant should be held upright over the shoulder and patted on the back until belching occurs. If this is ineffectual, a stomach tube may be inserted. Distention of the lower bowel is best relieved through the use of a rectal tube or an enema. A change of position of the baby often brings about the shifting of a large air bubble to a point where fewer symptoms are produced. The infant may be turned from side to side, held upside down, or placed on the abdomen. The application of external heat tends to relieve intestinal spasm. An effective method of treatment of the attack is to place the baby on his abdomen over a protected hot-water bottle. Carminatives, such as elixir of catnip and fennel (5 to 15 minims or 0.5 to 1 ml. in a little warm water), or a soda-mint tablet dissolved in a tablespoonful of warm water are of value. The symptoms of colic may be so severe that the use of sedatives is necessary in order to permit the infant to obtain some rest. Paregoric and phenobarbital are the sedatives most frequently used. Paregoric is effective in relieving the pain, but its use should not be repeated if it causes abdominal distention. The various measures mentioned are only palliative and temporary in their effect. The underlying cause of colic must be corrected.

In searching for the cause of colic, the character of the feeding should receive first consideration. If the infant has been underfed, a gradual increase in the amount of food should be made until an adequate diet is given. The first effect of the increased food intake may be an exacerbation of the symptoms of colic. These symptoms disappear, however, after a full diet is given and the infant has become accustomed to it.

It is bad practice to wean from the breast an infant who suffers from colic when the condition may be relieved by the giving of complementary or supplemental feedings or by regulation of the feeding regimen. In the breast-fed baby abdominal discomfort may arise from an overabundant milk intake, in which case lengthening the feeding interval is most effective if the interval is less than four hours. If this measure is ineffective, the giving of 25 to 30 ml. (one ounce) of boiled skimmed milk or a suspension of powdered casein immediately preceding the breast feeding may be tried. By this procedure less milk will be taken from an overabundant supply, and in addition the relative amounts of protein and sugar in the diet will be altered in the direction less favorable to intestinal fermentation. A similar procedure is useful also for the baby who is underfed at the breast, though if underfeeding is known to exist, a better-proportioned complementary feeding is preferable.

In the case of the artificially fed baby, the feeding of a suitable food mixture up to the point of satisfying fully the infant's appetite is usually a safe procedure and one that is unlikely to produce the symptoms of colic. When colic is present, one should be cautious about making frequent changes in the formula with the idea of decreasing the amount of some supposedly offending dietary component. Such a procedure too often results in decreasing the total food intake, thus aggravating the condition. The treatment of colic due to underfeeding consists in giving more food, not less. However, when colic is associated with evidences of excessive intestinal fermentation, it is

desirable to decrease the amount of added sugar or to change its type. Any decrease in the quantity of sugar should be compensated by increasing the quantity of milk so that the total energy intake will remain adequate. Also, in case of fermentation, the use of acidified milk may prove beneficial in decreasing bacterial activity. On the other hand, some infants have an excessive secretion of acid in the gastric juice and have abdominal discomfort when acid milk is fed. For these, sweet milk is indicated.

Some infants with hypermotility of the gastrointestinal tract suffer from hunger and abdominal discomfort when the feedings are given at four-hour intervals. In these the symptoms may be relieved by feeding more frequently. The intervals, however, should not be shorter than three hours.

Occasionally colic may be a manifestation of food allergy. In such case the baby may be benefited by the use of milk of which the proteins have been partially denatured by heat treatment, or it may be necessary to use a milk-free diet (see Chapter XVII).

Atropine is sometimes effective in the treatment of colic, especially when it is associated with gastroenterospasm. The method of administration is the same as that described in the discussion of pyloric stenosis (page 344). The administration of a formula thickened with starch is often useful in relieving the abdominal discomfort in cases of gastroenterospasm.

CHAPTER XXV

CONSTIPATION

Constipation results in the normal infant when the stimulus to peristalsis is insufficient or when the food residue is of such a nature as to form firm, hard masses that are expelled with difficulty. Constitutional and anatomical factors are of importance. Some intestinal tracts are active and easily stimulated to peristalsis; others are sluggish. Of two infants of the same age and given identical food, one may have several loose or semisolid stools a day regularly and the other a single hard constipated movement. Anatomical conditions, such as exceedingly long sigmoid colon or variations in the mesenteric attachments with resultant sagging or kinking of the bowel, predispose to stagnation. Infants with rickets and those who are anemic are likely to have poor muscular tone, a factor contributing to the development of constipation. Hypothyroidism is associated with sluggish intestinal peristalsis. Cretins usually are obstinately constipated.

Constipation associated with intestinal atony occurs when the diet is deficient in thiamine. However, it is probably seldom that thiamine deficiency of sufficient degree to produce constipation occurs in infancy. Many of the vitamin B preparations have a laxative effect irrespective of possible existing deficiency.

The normal stimulus to peristalsis is food. Any condition or circumstance leading to low food intake is likely to produce constipation. Pyloric stenosis or other condition associated with vomiting usually leads to constipation. When the food taken is so completely digested and absorbed that little residue remains in the bowel, movements

are infrequent; on the other hand if the residue left by the food is firm and nonirritating, this residue may accumulate in the lower bowel and be passed with difficulty. A food leaving a reasonable amount of soft residue is likely to lead to normal bowel evacuations.

The relative proportions of milk protein and sugar in the infant's food have a marked effect on the consistency of the stools and the frequency of their passage. In general, the sugars have a laxative effect, while milk protein, especially casein, tends to have the opposite result. Normally the effect of one of these factors tends to offset that of the other.

With increased sugar intake greater fermentation in the intestinal tract may be expected. The products of fermentation stimulate peristaltic activity. Lactose and sucrose are more laxative than more quickly absorbed sugars, such as dextrose. Although in general an excess of carbohydrate in the diet tends to cause diarrhea, one may encounter obstinately constipated infants who are receiving large amounts of such a carbohydrate as dextrin-maltose, maltose or dextrose.

Infants receiving considerable milk protein and little carbohydrate are likely to be constipated. With this type of diet the contents of the lower intestinal tract become alkaline. The alkalinity thus produced counteracts the laxative effect produced by fermentation of carbohydrate and also leads to the formation of relatively insoluble calcium soaps when fat is present in the diet. The soaps tend to form putty-like masses that are moved along the intestinal tract more slowly and passed less frequently. The addition of calcium salts in the form of lime water or other calcium preparations may be expected to increase the formation of calcium soaps and consequently to have a constipating effect.

Human milk contains relatively little casein in proportion to the amount of sugar present. It is for this reason

that constipation is less frequent in breast-fed infants than in infants fed the usual modifications of cow's milk. When constipation occurs in breast-fed infants, it is likely to be due to insufficiency of milk or to constitutional factors on the part of the infant. However, some breast-fed infants receiving adequate food and growing steadily are chronically constipated until supplementary foods are included in the diet.

Habit is an important factor in leading to constipation, especially in older infants. Unless measures are taken to encourage regular bowel evacuation by placing the infant on a vessel at certain times of the day, chronic constipation may result. The retention of fecal material in the lower intestine leads to dilation and atony of the bowel, with the result that the constipation is likely to become progressively worse. The pain of passage of a hard constipated stool, especially if a fissure is present at the anus, is likely to cause voluntary suppression of defecation.

Symptoms

The symptoms of constipation are few and are relatively unimportant except for fecal impaction with obstruction in instances of extreme and neglected constipation. The passage of large firm stools may in itself be painful; it may be painful also because of anal fissures produced by stretching. The existence of anal fissure is usually called to attention by the presence of small amounts of fresh blood on the outside of a constipated stool. Constipation has been assumed to lead to absorption of toxic products. However, the evidence is meager that such absorption occurs or that moderate constipation is in any way detrimental to health.

Treatment

In many instances of so-called constipation no treatment is required. If an infant is passing only one firm stool a day or even occasionally misses a day, but is thriving in

every respect, the character of the stools may be disregarded. If, however, the movements are passed with difficulty and accompanied by pain or if flecks of blood appear on the stool, treatment of the constipation becomes necessary.

Constipation will respond to dietary regulation in most instances. When caused by underfeeding, an adequate diet should be given, not so much to relieve the constipation as to provide for normal nutrition.

In constipation of artificially fed infants, who have been taking relatively large amounts of milk with little carbohydrate, the proportion of carbohydrate should be increased. Even when the formula proportions are such as are customary, a moderate increase in the sugar addition is often effective in relief. The sugar added should be one that is not absorbed rapidly. When sugar is absorbed, considerable water is taken with it, and if the amount of sugar is large and the absorption is rapid the stools may become even firmer. If the constipation is not relieved by an increased addition of the sugar already being used in the formula, a change to a more laxative type of carbohydrate addition is indicated (see Chapter IV). The most laxative of all the carbohydrate preparations are the liquid malt extracts, particularly those to which potassium carbonate has been added. From 15 to 50 ml. ($1\frac{1}{2}$ to $1\frac{1}{2}$ ounces) may be included in the total day's feeding and, according to the effect produced, may either replace an equivalent amount of other carbohydrate or may be given in addition to the carbohydrate already present in the formula. Usually replacement is all that is necessary. The amount given should be regulated by the effects produced.

The constipation of artificially fed infants may be relieved further by removal of a portion of the fat of the milk in order that a smaller residue of calcium soaps may

remain in the bowel. Usually it is not necessary to resort to this form of diet modification.

One should always bear in mind that the condition of the infant and not that of the stools is the primary consideration. It is proper to alter the diet in order to relieve constipation provided the alteration is not of such type as to make the feeding inadequate to meet the nutrition requirements.

In the case of breast-fed infants little or nothing can be done to alter the composition of the milk, nor would one know just what alterations would be desirable, even if possible. It is important that the total quantity of milk ingested be adequate for good nutrition. When all the nutritional factors seem satisfactory, the simplest effective means of relieving constipation is the giving of liquid malt extract in a little water preceding one or more of the breast feedings. For infants four or five months of age or older, fruits and vegetables are useful, as is discussed subsequently.

Some of the fruit juices have a moderate laxative effect. Orange juice has little influence in relieving constipation; prune juice, on the other hand, is fairly effective.

Normally, sieved fruits and vegetables may be added to the diet at four to five months of age (see Chapter XVIII). These foods have some laxative effect, largely because of the unabsorbable residues. For babies who are constipated, fruits and vegetables may be given somewhat earlier than is commonly customary, or, if they already are being given, the quantity may be increased. Oatmeal and other whole grain cereals are more laxative than those which have been highly milled.

Though thiamine has little laxative effect except when constipation has resulted from thiamine deficiency, various vitamin B complex preparations often are markedly laxa-

tive. This effect possibly may be dependent on materials other than the vitamins present. Dextrin-maltose preparations to which extracts of yeast or wheat embryo have been added may serve well in the relief of constipation when used as the added carbohydrate in the preparation of milk formulas; or the vitamin B complex preparation may be given independently and not as a part of the formula.

In all cases of constipation the institution of regular habits is important. In the latter half of the first year many infants may be trained to pass the stools at regular times. Passage of stools at scheduled times is encouraged by gentle massage of the abdomen, following along the direction of the colon. This type of prescription is of special value in infants with lax abdominal walls.

Mechanical means of emptying the bowel may be required occasionally. A soap stick, greased paper cone, or suppository may be used to encourage regular habits and temporarily to relieve constipation. Glycerin suppositories are irritating and should not be used regularly, although no serious objection to occasional use exists. Enemas also should be used only as an occasional and temporary measure. The enema habit is almost as bad as the cathartic habit.

When the simple measures that have been indicated are used, the need for recourse to drug treatment will rarely arise. The habitual use of cathartics is to be condemned. However, if other means of treatment fail, resort to drugs may become necessary. Mineral oil is commonly used, but it is not to be recommended. It acts by keeping the stools soft, but also it causes loss from the intestinal tract of important amounts of carotene and vitamin A. A preferable preparation for keeping the stools soft is a dried extract of psyllium seed. The powder may be moistened and fed with a spoon. It acts by absorbing and holding water.

Milk of magnesia has been used widely for the relief of constipation in infants. It is partly converted into magnesium chloride in the stomach and its action is the same as that of magnesium sulfate, that is, as a hydragogue laxative. In the relatively small amounts usually necessary it is entirely harmless.

When blood-streaked stools indicate the presence of anal fissures, the stools should be kept soft by the use of mild laxatives, if necessary, for at least ten to fourteen days until the fissures have healed. Application of 5 per cent silver nitrate solution to the fissures promotes healing.

CHAPTER XXVI

ALLERGY

It is not proposed in this chapter to present a full discussion of the subject of allergy, but only to consider certain phases of allergy which have a direct bearing on infant feeding.

The term allergy is used to designate an abnormal or altered degree of sensitivity to antigens. An antigen is any substance that can cause the production of antibodies to *the antigen*. Antibody is present usually as a result of previous introduction of antigen. When further antigen is introduced a symptom-producing reaction occurs. When the allergic state develops as a result of previous introduction of antigen in a person whose capacity to become susceptible is not an hereditary constitutional trait, the allergic response to further introduction of antigen is known as anaphylaxis. The term atopy is often used synonymously with allergy, but its use is more correctly restricted to certain allergies present in those with an hereditary constitutional capacity to develop allergy. In the restricted atopic group belong hay fever, asthma, some urticarias, and certain of the eczemas. Asthma and hay fever are rare in infancy and urticaria is uncommon. A very rare atopic phenomenon is sudden death as the result of introduction of the antigen, but it is an event that must be kept in mind in the presence of known atopy.

It is seldom that inhalants are responsible for allergic response in infants; symptoms from inhalants appear later. In most instances in infancy the allergy is to ingested antigens; in a few, it is to materials injected parenterally for prophylactic or therapeutic purposes, as, for example, foreign serum. In rare instances violent reactions occur with

the first ingestion or injection of the offending antigen. It is not clear how such constitutional atopy arises, although it is possible that in some instances sensitization of the fetus occurs during intrauterine life as the result of passage of proteins or antibodies from the maternal to the fetal blood.

In postnatal life, sensitization may occur from ingested foods. During early life very small amounts of undigested protein pass through the intestinal mucosa directly into the blood stream. Some proteins are more likely to do this than others, especially egg white. In the presence of gastroenteritis, larger amounts of food protein may be absorbed unchanged. It has been claimed that even human milk may contain foreign food proteins which have formed a part of the mother's diet, and in this way human milk may sensitize the infant. Cow's milk protein, being from a different species, is far more likely to lead to sensitization. Of the proteins of cow's milk, the lactalbumin is a more frequent offender than the casein, probably because unlike casein it is not coagulated during the processes of digestion and hence is more easily absorbed. All cereals contain some protein, and the proteins of wheat are especially likely to act as allergens.

It is recognized also that substances other than proteins may give rise to symptoms indistinguishable from those of protein allergy. Drug idiosyncrasy may be of the same nature as allergy. Even the application of physical agents, such as heat, light or cold, may lead to allergic reactions in susceptible persons. The effect of the offending allergens may be observed when the substances in question are administered by mouth, subcutaneously or intravenously, when they are inhaled or when they merely come in contact with the unbroken skin.

From the standpoint of infant feeding, the principal concern is with the allergic reactions which possibly may be the result of the ingestion of those food materials which

ordinarily make up the infant's diet. Of the various constituents of the diet, the proteins are most likely to cause reactions, although occasionally other food constituents, such as oils, may be responsible.

In rare instances, violent reactions occur when even minute amounts of the offending allergens are fed; thus an infant, after taking a single swallow of cow's milk, may develop angioneurotic edema, urticaria, dyspnea, and profound shock. Minor manifestations of allergy are more frequent. It is possible that allergy may be the underlying cause of a great variety of minor disturbances in infants. Often a tendency is encountered to attribute almost any bizarre symptoms to allergy, but either direct or fairly strong circumstantial evidence exists that allergy plays a rôle in the causation of several common conditions. It is not to be assumed that allergy is the only cause of the symptoms observed, for it is often only one of a number of causes which may lead to similar manifestations. When other possible causes have been eliminated, it may be well to consider allergy.

Among conditions in infancy which *may* have an allergic basis are to be included urticaria, eczema, colic, gastroenterospasm, vomiting, diarrhea, recurrent bronchitis and chronic rhinopharyngitis. Certain of the manifestations of the so-called "exudative diathesis" and "neuropathic diathesis" may in reality be evidences of an allergic disturbance.

It is not always possible to prove that allergy is the cause of the symptoms, or to identify the offending protein. The usual skin tests cannot be depended upon absolutely. A fair proportion of apparently normal infants give positive skin reactions to milk and other proteins, and other infants who appear to be sensitive to certain food proteins may not give positive skin reactions to the proteins in question. The passive transfer method of skin testing is

sometimes of value when the usual skin tests fail, but it rarely gives much information in the case of infants.

In order to make the diagnosis of food allergy or to determine which foods are the offenders, it is often necessary to resort to an empirical elimination process. By one method of procedure the various protein-containing foods entering into the diet—milk, cereal, eggs, fruits and vegetables—are omitted one by one and then later added. If the symptoms disappear when the food is withdrawn and reappear when it is again introduced into the diet, it may be suspected reasonably that the food in question is the offender. This plan is not applicable when multiple sensitivities to foods exist. Consequently, a procedure with wider application is to devise a basic simplified diet to which the baby is not likely to be allergic. If the choice of foods has been correct, the allergic symptoms should disappear. Subsequently the foods customarily prescribed for infants are added one at a time. If a reaction occurs, the newly added food is withdrawn and added again at a later time for confirmation of its responsibility for the reaction. The process of carrying out this type of regimen is long and tedious, but ultimately the offending foods will be determined with considerable accuracy and the diet will approach closely one that is customary.

In devising the basic elimination diet either milk or a milk substitute must be used. In addition, all the known nutritional essentials not supplied by the milk or the milk substitute must be given to make the diet complete. For this purpose use may be made of the vitamin concentrates or crystalline vitamin preparations instead of their food sources, and of iron salts instead of food iron. Other mineral additions may be needed. The milk preparation chosen should be one that has had its proteins at least partially denatured. The heating of milk denatures the protein to an extent depending on the degree of heat and the length of time it is applied. Boiling of milk for many

hours (with reflux condenser) denatures the protein to a considerable extent. Autoclaving the milk, as is done in the manufacture of evaporated milk, denatures the protein to some extent. Acidification of milk partially denatures the casein, but has little or no effect on the lactalbumin. If these forms of cow's milk do not prove satisfactory it may be necessary to eliminate cow's milk entirely from the diet. As a substitute, goat's milk may be tried, but infants who are sensitive to the casein of cow's milk are also often sensitive to the casein of goat's milk, though they may not be sensitive to the lactalbumin.

If neither cow's milk nor goat's milk proves satisfactory, it may be necessary to eliminate milk entirely from the diet. Infants may be fed a milk-free diet if it is properly constructed. One of the most used substitutes for milk protein is the protein of the soybean. Several soybean preparations intended exclusively for this purpose are commercially available. To these, certain additions have been made in order that the foods will be more nearly complete. When these preparations are used care must be taken to make the diet complete and to give the infant sufficient calories. The necessary supplements include iron, vitamins A, C, D, and those of the B complex. Another milk substitute has hydrolyzed casein for basic content, with appropriate carbohydrate and mineral additions. This preparation must be supplemented with vitamins A, C, D, and those of the B complex.

When some specific food such as wheat, oats, rice, tomatoes, orange juice, or cod-liver oil appears to be responsible for the symptoms, that particular food should be eliminated from the diet and, if indicated, the equivalent food value should be given in other ways. When foods are eliminated from the diet because of allergy, it is desirable at all times to keep in mind that the diet must be kept complete. Too often nutritional deficiency diseases have been permitted to

develop because of the dietary restrictions incident to the prescribing of a nonallergenic diet.

Babies usually may be desensitized to those proteins causing allergic symptoms. Often desensitization is unnecessary because the food responsible is not nutritionally important. Milk is so important in the diet of the infant that desensitization is desirable. It may be accomplished by giving first minute amounts of milk, and then gradually increasing the amount day by day until appropriate amounts are being taken without reaction.

A condition attributable in part to allergy and occurring fairly commonly in infancy is eczema. Allergy appears to be a large factor in the production of infantile eczema in many instances, but existing evidence does not support a contention that all eczema of infants is dependent on this cause. Opinions differ widely as to the importance of allergy in the causation of eczema. Allergy may be a cause in possibly not more than 25 per cent of the cases. Even when the eczema is an allergic manifestation, the sensitivity is by no means always to food. Environmental factors, such as feathers, may be responsible in as many as one-third of the instances of allergic eczema in babies. When allergy to food is the cause, dietary changes are important in the management. In many instances it is not easy to determine quickly whether the eczema is on an allergic basis or to identify the responsible allergens. Consequently elimination diets are used frequently in the treatment of babies with eczema irrespective of the finally determined cause. Fully as important as the dietary changes is the appropriate use of local applications to the affected areas of skin. In many instances environmental changes also are highly important.

An elimination dietary regimen is carried out more easily in a hospital than in the home. When it is tried in the home, the keeping of a food diary by the mother is often of great assistance to the physician. The relationship of

food ingestion to exacerbations and remissions of the eczema is more definitely determined by means of the recorded diet and data than by the often vague impressions of the mother.

Gastrointestinal allergy is not common in infants, but its recognition is important when it occurs. The diagnosis is difficult to prove, but the evidence is satisfactory if withdrawal of any food relieves the condition and ingestion of the food causes recurrence of the symptoms. Vomiting tends to occur only when the allergy is severe. The more common symptoms are colic and diarrhea. The stools contain much mucus, and blood may be present in small or large amount. Also cases have been reported in which the symptoms resembled somewhat those of celiac disease, presumably because of edema of the intestinal mucosa and failure to absorb fat. However, in celiac disease utilization of complex carbohydrate is also affected.

CHAPTER XXVII

PREMATURITY

Infants born before the thirty-sixth week of gestation and those weighing less than 2500 grams (5½ pounds) usually differ anatomically and physiologically from normal full-term infants. Low birth weight is not necessarily the result of premature birth, as the small size may be an inherited characteristic; nor do all infants who have been born prematurely necessarily weigh less at birth than the average for infants born at term. In general, however, those infants who are born prematurely and those who are very small at birth require special care and feeding. In those instances in which the premature birth is the result of disease, such as syphilis, further special therapeutic measures may be necessary.

Prematurity and the Newborn Period

All studies of the newborn period tend to show that adaptation of the normal full-term infant to independent life is not an immediate performance, nor yet a matter of a few hours, but requires days or weeks to achieve. When a baby is born prematurely, it is to be expected that immaturity of function is of greater degree than in the full-term infant, and that maturity of function is more slowly attained. Survival of the prematurely born infant depends in large measure on the ability of his custodians to compensate for his immaturity. Knowledge of the degree of functional maturity of the infant is therefore a prelude to intelligent care.

Respiration.—Establishment of respiration is a prime essential to survival of the newborn infant. During fetal life the oxygen supply from the placenta is sufficient for

normal development of the tissues. Extrauterine respiration is initiated by anoxia. If the period of anoxia is unduly prolonged from any cause, such as prolonged labor, excessive anesthesia or narcosis, damage to the respiratory center may be so great that it functions incompletely or not at all. The newborn infant, mature or immature, can survive a longer period of anoxia than the average adult, yet the longer the period of anoxia, the greater the possibility of irreparable damage to the central nervous system. Conversely, intracranial hemorrhage may cause sufficient damage to the respiratory center to produce apnea in the infant.

The muscular effort required to start breathing is greater than that needed to keep it going, but usually is well within the ability of even the prematurely born infant. Expansion of the entire lung may take a week or more in the full-term infant; the weaker immature infant may not be able to expand the lungs completely for a still longer period. Breathing in the newborn is always diaphragmatic and abdominal. The intercostal musculature is weak.

Because of the small size of the infant, relatively more air must be moved in and out of the lungs than for the adult; this is accomplished by a much faster rate of breathing, rather than by deeper respirations. The mature newborn infant breathes approximately 44 times a minute, the immature still more rapidly; the tidal air is only about 20 ml. even in full-term infants. Breathing may be regular, irregular, or periodic in the full-term infant. Irregularity in breathing rate is common in immature infants. Particularly in the immature baby periods of regular or irregular breathing may be interrupted by long periods of apnea, sometimes leading to death from asphyxia. Apnea in prematurely born infants may be caused also by intracranial hemorrhage affecting the respiratory center; in fact, hemorrhage is more often the cause of apnea than is immaturity of the nervous system.

Intracranial hemorrhage occurs at birth more frequently in babies born prematurely than in those born at term. The reasons for this difference pertain to the stage of development. Most of the calcium present in the body of the full-term infant at birth is deposited during the last three months of fetal life, and one-half of it during the last month. The cranial bones of the baby born prematurely are much softer and more pliable than those of the baby born at term. It is usually the head that dilates the birth canal. When the cranial bones are soft, marked molding of the head is almost certain to occur and tears of the falx or tentorium are much more likely. It is such tears that produce hemorrhage. In addition, the capillaries of the immature infant rupture with much less pressure than do those of the full-term newborn infant. Thus, intracranial hemorrhage is common despite "normal" delivery or birth.

Circulation.—The relatively large surface area of the newborn infant makes necessary an output of blood from the heart of about 500 ml. a minute, an amount greater in proportion to body weight than that for the adult. In relation to the total size of the baby the heart is relatively large at birth, with a maximum diameter of 5.5 cm. (2.5 in.). Cardiac murmurs occur in a considerable proportion of newborn infants without permanent cardiac abnormality. On the other hand, infants without significant murmurs at birth may develop them later.

The pulse rate, like the respiratory rate, is most variable during the neonatal period. Values from 90 to 180 a minute are not unusual for brief periods, though 125 to 130 a minute is the more common finding. Blood pressure determination, to be accurate, should be taken with a narrow cuff. A suitable width is 2.5 cm. (1 in.) for full-term infants, narrower for those born prematurely. Systolic pressures of prematurely born infants are 60 to 80 mm. of mercury; those for full-term infants increase from about

80 at birth to 95 or 100 by two weeks of age. Circulation at the periphery is less well developed than that of the viscera. The number of skin capillaries is relatively small.

Heat Regulation.—During fetal life heat production is low; it still is lower in the newborn infant than in the older infant or child. The heat production of the prematurely born infant, who leads an essentially vegetative existence, with little muscular activity, is less than that of the full-term infant.

Not only is heat production low, but the mechanisms for conservation of such heat as is produced are poorly developed at birth. The surface area is relatively large and the subcutaneous fat is small in amount. The skin is poorly supplied with capillaries and its vasomotor control is poor. The sweat glands and the mechanism for shivering function poorly in the newborn infant and not at all in the immature. Prematurely born infants show great thermolability. The body temperature tends to fall below normal on slight exposure to cold and to rise above normal when the environmental temperature is high. Daily variations in temperature of as much as 3° C. (5° F.) may be observed when the environment has not been regulated carefully.

Digestive System.—Development of the secretory glands and absorptive surfaces of the gastrointestinal tract has proceeded further at birth than the development of its supporting musculature. The amount of free hydrochloric acid in gastric juice of the newborn infant is as high as in the adult; however, the amount of acid decreases by the third day and remains below the adult level during infancy. A large proportion of immature infants are achlorhydric. The digestive enzymes are usually present in adequate amount with the exception of amylase. Fat is often poorly handled by young infants, especially by those born prematurely. Olive oil seems to be better utilized than butter fat. Some immature infants apparently do not digest or

absorb fat at all; such babies will fail to gain weight with an apparently ample intake. Prematurely born as well as full-term infants can digest and absorb protein in larger amount than is usually fed.

Gastrointestinal motility is poor even in the full-term newborn infant, much more so in the immature. The gastric capacity of the immature infant is likely to be disproportionately small. The presence of swallowed air in the intestines delays absorption. The nutritional requirements of the prematurely born infant cannot be estimated on the same basis as those of the average-sized young infant. During the first two or three weeks after birth the energy requirement is low in proportion to the body weight, largely because of the small amount of muscular activity. On the other hand, absorption from the gastrointestinal tract is likely to be poor. As a result of the combination of these two factors, the energy requirement is about the same as for the full-term infant. The energy requirement for the first two or three weeks varies from 100 to 120 calories daily for each kilogram (45 to 55 for each pound). After the first few weeks the energy requirement increases rapidly as the infant becomes more active; it then usually exceeds that of the full-term infant and may be as high as 155 to 175 calories daily for each kilogram (70 to 80 for each pound). Relatively large amounts of protein and mineral salts are required because of the rapid rate of growth. Some of the vitamin requirements also presumably are higher than for the baby born at term.

The increased susceptibility of the prematurely born baby to rickets is well known, but rickets, when it occurs in the immature baby, is dependent as much on rapid growth and low intake of mineral as on deficiency of vitamin D. If the baby is born a month prematurely, the skeleton contains only about one-half as much mineral as that of the full-term baby at birth. In order that the bones may grow in a normal and expected manner it is necessary

that calcium and phosphorus be added to them at a rate much greater than is necessary for the skeletal growth of the full-term baby. Under the best of circumstances only a portion of the mineral that is ingested is retained. When human milk constitutes the sole source of mineral, the amount of calcium ingested by the prematurely born infant usually does not exceed that which he should retain to have normal growth; it often does not exceed that which is necessary for maintenance before retention begins. In the past, the feeding management of prematurely born babies often has been characterized by lack of recognition of the mineral need and failure to supply it. Because rickets developed in these circumstances, excessive amounts of vitamin D have been given.

Urinary Excretion.—The kidneys of the full-term infant at birth are functionally immature; those of the immature infant may be anatomically immature. Functional maturity of the kidney is not attained for many weeks after birth. On this fact depends many of the disturbances peculiar to immature and full-term infants.

The newborn infant is unable to concentrate urine to the same degree as the older child and adult. Thus, more fluid is required to carry out a given amount of solids. During the first day or two after birth the water intake is often low and nitrogenous waste accumulates in the blood. Clearance tests show decreased ability to regulate excretion of both salt and urea; albuminuria is a common finding in normal newborn babies and almost a constant finding in those who are immature. The decreased ability of the kidneys to regulate salt metabolism is responsible for the hydrolability of the newborn and especially of the prematurely born infant.

Acid-Base Equilibrium.—The blood of a normal prematurely born infant contains a relatively large amount of organic acids, chiefly pyruvic and lactic acids, with ketones low or absent. In illness the organic acid content

risers alarmingly. As pyruvic and lactic acids are products of incomplete combustion of carbohydrate, it appears that the organic acidosis of immature infants may be dependent on incomplete oxygenation in tissues. Since all prematurely born infants are slightly acidotic to begin with, any considerable amount of diarrhea or dehydration can result in acidosis of much more critical degree than would be the case with older infants.

The Management of the Prematurely Born Infant

In the management of the prematurely born infant factors listed in the preceding discussion must be given special consideration. The body temperature must be maintained at a relatively normal level, infections must be avoided, and respiratory activity must be maintained. The diet must be sufficient to cover the special requirements and yet must be small in volume and easily digestible. Careful and intelligent nursing is essential.

In order to maintain the body temperature the prematurely born infant must be clothed properly, kept in a warm environment, and not exposed to the chilling effects of injudicious bathing. The clothing should be so designed as to provide against loss of body heat, but at the same time should be arranged for ready changing without subjecting the infant to undue chilling. Suitable clothing consists of the customary shirt and diaper and a quilted cotton jacket such as shown in Fig. 9. This jacket is made from cotton batting covered with gauze and is provided with a hood to cover all the baby's head except the face. The jacket should be sufficiently long to extend at least six inches below the baby's feet and wide enough to envelop the infant completely and to lap over several inches (Fig. 10). The jacket is opened but not removed when the diaper is changed. The diaper should be easily removable and not fastened with too many pins. The jacket usually is unnecessary if the infant is kept in a special incubator room such as is provided by some hospitals.



Fig 9.—Premature jacket of quilted cotton and gauze.



Fig. 10.—Premature jacket applied to infant.

The infant not only should be protected by suitable clothes, but also should be kept in a warm environment. This may be accomplished either by putting the infant in a small warm room or in one of the several varieties of heated infant beds or incubators. Whatever the means employed, it is desirable not to maintain the body temperature at a level higher than 98.6° F. (37° C.) A body temperature one degree below normal is preferable to a temperature one degree above normal.

The temperature of the warm room should be 85 to 90° F. (29 to 32° C.) for very small infants, and 80 to 85° F. (26 to 29° C.) for those weighing over four pounds. The temperature of the room should be regulated to that which is necessary to maintain the baby's temperature at a suitable level. Fresh air is essential but drafts are to be avoided. The air of the room should be moist, a relative humidity of 55 to 60 per cent being appropriate under average circumstances. Suitable humidity of the air is fully as important as the maintenance of the environmental temperature. Incubator rooms of some hospitals are provided with special, often automatic, means of maintaining humidity. In other circumstances it may be maintained by evaporating water from an open basin on an electric stove or by placing pans of water or moist cloths on a radiator. The degree of humidity may be determined by the use of humidity-indicator instruments or by the use of the wet and dry bulb thermometer.

It is not always possible to provide a suitable warm room which may be maintained at a constant temperature and be properly ventilated, nor is such a warm room absolutely essential. The baby may be placed in a heated bed improvised from a clothes basket lined with quilting and kept warm by the use of hot water bottles or well-protected electric pads. Considerable skill and constant attention are required in keeping the temperature just right by these means, and danger of overheating or burning the infant

exists. With careful supervision, however, satisfactory results may be obtained.

Various incubator beds with automatic heat control have been devised. Of these, a satisfactory and widely used variety is the Hess incubator bed. The temperature of the bed usually is kept at about 85° F. (29° C.). The chief disadvantage of many incubator beds is the lack of means for providing humidity. Some incubator beds have such a provision.

When the baby is kept in a suitably warmed room, he can be removed to a dressing table, if desired, to have various offices performed for him. He should not be removed from the warm room or from the incubator bed for bathing and changing. A soap and water bath usually is considered inadvisable for the very small baby. In cleansing the infant, only a portion of the body should be exposed at one time and cottonseed or olive oil should be used instead of water. Desirable though it may be to know the weight and progress of gain of the infant, it is inadvisable to make daily weighings, as this involves considerable exposure—unless it can be done in an incubator room with special precautions. Weighing every three or four days is sufficient. The infant should be wrapped in a warm blanket for weighing, the weight of the blanket being deducted subsequently from the total weight.

Prematurely born infants are extremely susceptible to infections, and even such a mild infection as a cold may have fatal consequences. Whenever possible, only one nurse should be in charge of the infant, with a single individual to relieve her. As an extra precaution against infection the nurse should wear a face mask when handling the infant. Should the nurse contract a cold or any other infection, she should be relieved from duty promptly. No visitors should enter the room. Maintenance of appropriate humidity in the room decreases somewhat the infant's susceptibility to infection.

The respiratory effort of the prematurely born infant at best is poor, and often is insufficient to expand the lungs completely. The respirations are not only shallow but often irregular. At times it appears that the infant simply "forgets to breathe." During these apneic attacks cyanosis may become extreme and death may occur. The maintenance of normal respiratory activity is difficult, as the respiratory center is relatively insensitive. Sensory stimulation of the skin is impractical as these infants must be kept warmly clothed and not subjected to chilling. The most effective means of bringing about expansion of the lungs is inhalation of a mixture of oxygen and carbon dioxide. These mixtures may be obtained in small cylinders. Either a mixture containing 5 per cent carbon dioxide or one containing 30 per cent may be used. The 30 per cent mixture has a strong stimulating effect on the respiratory center and should be used for only short periods when the respiratory action has practically ceased. The inhalation of a 30 per cent mixture for periods of from 3 to 5 minutes every three or four hours serves to expand the lungs and to prevent atelectasis. The 5 per cent mixture is suitable for prolonged inhalations after the respirations have been started. The inhalation of these mixtures brings about an increased carbonic acid content of the blood without diminishing the oxygen content.

Inhalations of oxygen alone are also of value in relieving cyanosis. In giving the inhalations, a bottle of water with the inlet tube reaching to the bottom should be connected between the tank and the funnel or catheter to be used. The tank valve should be so regulated as to allow from 60 to 80 bubbles a minute to pass through the water (Fig. 11). The oxygen may be administered to the baby by means of a funnel or small face mask, or by a small-sized nasal catheter. The latter method is more effective and economical of oxygen, but is open to the objection that any sudden change in the pressure of the gas may damage

the infant. The catheter method should not be used unless the gas tank is supplied with a high grade valve capable of fine adjustment. An oxygen tent may be used, but often it is not easily adapted to the other requirements in the care of the infant. Some incubators are provided with an oxygen inlet.

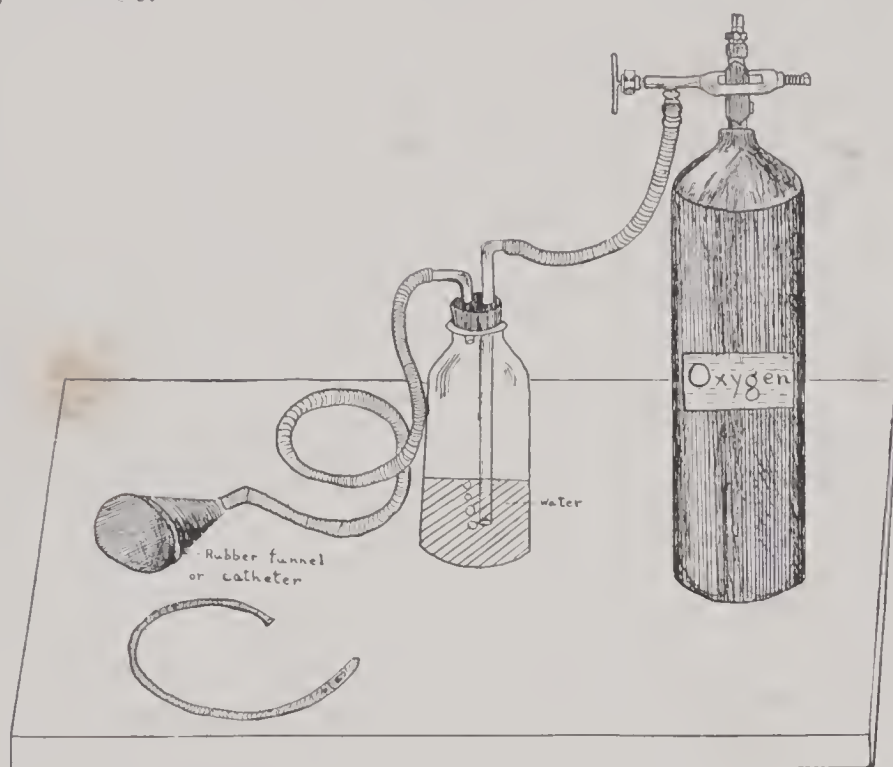


Fig. 11.—Oxygen inhalation apparatus.

The sprinkling of a little cold water on the infant's face sometimes serves to stimulate respiration, as does also the administration of drop doses of aromatic spirits of ammonia. Alpha-lobeline is sometimes used as a respiratory stimulant, but, in overdosage, it is capable of producing respiratory depression, vomiting, and collapse. The initial dose, given subcutaneously, should not exceed $\frac{1}{40}$ to $\frac{1}{20}$ gr. (0.0015 to 0.003 Gm.) and this should not be repeated within four to six hours.

The feeding of prematurely born infants presents special difficulties. The digestive capacity is small, and only small volumes can be given at a time because of the small gastric capacity, yet the total food requirement is fairly high.

Feeding at short intervals is likely to lead to vomiting. Some infants may be fed every two hours, but, for the majority, three- or even four-hour feeding intervals are preferable.

For the earliest feedings of the infant born prematurely, the food of choice is human milk because of its easy digestibility. The infant is too weak to suckle vigorously and it is generally necessary to obtain the mother's milk by manual expression or by means of a pump. Because of premature delivery, the mother is likely to have little milk. If insufficient milk is obtained from the mother, the milk of another woman may be used.

For the average prematurely born baby the customary regimen is to offer nothing by mouth during the first 12 hours after birth. After 12 hours a small quantity of water (4 ml.) is offered in order to determine the reaction of the baby to feeding. Subsequently human milk is given at the chosen intervals in quantities of 4 ml. or more, depending on the size and reaction of the infant. During the second 24-hour period the amount of milk at a feeding is increased to 8 to 20 ml., and on the third day another slight increase may be made. During these first three days it is customary also to offer water in quantities equivalent to the amounts of milk that are being given, the water being administered halfway between the feedings of milk. In order that both these materials be given satisfactorily, the feeding interval for the milk cannot well be shorter than three hours. A fairly good general rule for the prescribing of human milk is to give it in a quantity equivalent to one-twentieth of the body weight on the first day that milk is fed; thereafter the quantity is increased gradually until it is equivalent to one-fifth of the body weight. In the case of the average baby, after the third day it becomes desirable to fortify the human milk (as discussed below) and to decrease the amount of water offered between feedings.

When babies are extremely small at birth (1200 grams or less), no food should be given in the first 36 to 48 hours.

During this period the water content of the body should be maintained by hypodermoclysis; the quantity of fluid desirable is from 10 to 20 mil., given twice daily.

If the infant is able to take a sufficient volume of human milk in the course of a day, his energy requirements may be met. However, this is not always possible, as some infants will take no more than half an ounce or an ounce at a feeding, and will vomit when fed more often than every three hours. In such instances fortification of the milk becomes necessary in order that an adequate energy intake be obtained in a small total volume. Even when the energy requirements are met by means of human milk, the requirements for protein, calcium, and phosphorus usually are not met fully. The rate of growth expected of the baby up to the time he is the equal of a baby born at term is more rapid than can be accomplished satisfactorily with the amounts of protein and mineral in the quantity of human milk he can ingest. Consequently, fortification of human milk becomes desirable in order to satisfy the nutritional requirements other than of energy.

For the first few days—or few weeks, depending on circumstances—the primary consideration is survival. For this purpose human milk is best suited, because of its easy utilization. Subsequently normal growth is desired, as well as survival. It is largely because of failure to supply these needs adequately that a special susceptibility to rickets and anemia is attributed to babies born prematurely.

A material commonly used in the fortification of human milk is dried skimmed milk, though fresh boiled skimmed milk or acidified skimmed milk may serve as well. Prepared casein also is used for this purpose. One may add safely as much as 2 per cent by weight of casein, or 5 per cent of dried skimmed milk.

When human milk is not available, some type of cow's milk formula is used. Simple dilution formulas, evaporated

milk mixtures, or acid milk formulas may be prescribed according to the principles discussed in Chapters XIV, XV and XVI. Since fat is more likely to cause digestive disturbance than the other constituents of milk, low fat milk is to be preferred. Corn oil has been shown to be more easily utilized by the prematurely born baby than cow's milk fat. Because of this fact, a proprietary food has been devised especially for the feeding of these babies; in this food corn oil replaces the butter fat; and calcium caseinate, a dextrin-maltose mixture, and vitamin A have been added. Sometimes protein milk with 6 per cent added sugar is used, this mixture being relatively low in fat and adequate in most other respects. The protein milk may be given undiluted except during the first few days after birth; if dried protein milk is used, it is reconstituted to its original volume.

Mechanical difficulties are likely to be encountered in the administration of any type of feeding. Most prematurely born babies will not suck well on a nipple, and furthermore the usual-sized nipple is too large for the mouths of very small infants. Special types of feeders have been devised, such as the Breck feeder, but these devices are difficult to keep clean and possess no advantages over a medicine dropper or a rubber bulb. If a medicine dropper is used, the tip should be protected by a short length of narrow rubber tubing. The bulb often employed for feeding is an all-rubber bulb of the type sometimes used for syringing the ears. In using either of these devices, small amounts of milk are allowed to flow into the infant's mouth at a time. If the infant swallows well, this method is satisfactory, but some infants will hold a portion of the milk in the mouth and allow it to flow out, or they may aspirate some of it. In such instances resort must be had to tube feeding.

In feeding by tube, a small catheter, not larger than No. 12 French, should be used. This is inserted through the mouth or nose and passed down to a point just above the cardiac orifice of the stomach. It is better not to allow the catheter to enter the stomach, where it may cause irritation.

The length of catheter to be inserted will vary, of course, with the size of the infant. For an infant weighing 4 pounds (1800 grams), the length of the catheter to be inserted, measured from the margin of the gums, will be 5 to 6 inches (12 to 15 cm.). This point may be marked with indelible ink on the catheter. Milk should be allowed to flow into the stomach slowly by gravity from a glass tube receptacle held not more than 6 to 8 inches (15 to 20 cm.) above the head. After feeding, the catheter is pinched off before withdrawal so as to prevent dripping of milk into the pharynx. Tube feeding may be accomplished with little disturbance to the infant. In fact, some infants sleep through the entire procedure.

The quantity of food to be given for the first few days after birth already has been discussed. For the subsequent feeding the same general principles hold. No set rule may be laid down as to the volume of food which the infant of a given weight may be expected to take at a feeding. As more food is needed, the amount should be increased cautiously. The regular occurrence of vomiting after a feeding is an indication that too large a volume is being given. Vomiting is particularly dangerous for the prematurely born infant because of the likelihood of aspiration of the vomitus. Because of the small volumes which it is possible to feed at a time, frequent feedings have been advised in order to give sufficient food in the course of a day, but in general the better method of introducing more food is to give more concentrated feedings at longer intervals. The four-hour interval may be used to advantage for many infants, and it is rarely necessary to feed more often than every three hours. In every individual instance, the feeding intervals and volume of the feedings must be regulated according to the response of the infant.

The symptoms of vomiting and diarrhea are to be managed along the same general lines as indicated in the case of infants born at term. When vomiting occurs, the volume of the feedings should be decreased and the feeding in-

tervals, if short, should be lengthened. In order to prevent inadequate food intake, the food should be made more concentrated. Prematurely born babies become dehydrated easily and quickly unless precautions are taken to insure adequate hydration. With even moderate dehydration vomiting is likely to occur as a symptom, and the vomiting, in turn, increases the dehydration. Maintaining a high relative humidity in the environment assists greatly in preventing dehydration and its associated vomiting.

Diarrhea is always serious in the case of prematurely born infants, and severe diarrhea usually is fatal. The treatment is rendered difficult because these infants cannot be starved for long. Food must be supplied in some form even though diarrhea persists. Certain alterations in the type of food may be indicated. If human milk is being used, it may be acidified with lactic acid, using two drops of acid to the ounce of milk. If an artificial feeding is being used, a portion of the fat may be removed. Dextrose may be used in place of any other added sugar. Parenteral administration of fluid often becomes necessary to prevent dehydration. Other measures discussed in the chapter on Diarrhea may be indicated at times.

The amount of vitamin D required by the prematurely born infant is discussed in Chapter VII. While current opinion supports the administration of relatively large amounts of vitamin D to these immature babies, no satisfactory evidence exists that the requirement is greater than for infants born at term. Because of the unknown possibility of a greater need, more may be given, but it is believed that 800 units daily is fully adequate. Since it is possible to obtain this amount of vitamin D in a single teaspoonful of high-potency cod-liver oil, it is possible to supply the need of the infant from this source. If cod-liver oil is used, a divided dose is desirable in the early weeks. Because of the danger of aspiration, with subsequent serious lipoid pneumonia, many physicians prefer

to give vitamins A and D to the prematurely born infant in the form of a water-miscible concentrate, which may be added directly to the food.

The need of the infant for vitamin C is fully as great as is that of the baby born at term (p. 124). Strained orange juice may be given in small and increasing amounts from the earliest weeks. If desired, dissolved ascorbic acid may be added to the milk immediately before feeding.

The prematurely born infant tends to become anemic unless special precautions are taken (see p. 88). Considerable amounts of blood must be formed as the baby grows, and the stores of iron in the body are relatively small. After the first few weeks the addition of small amounts of iron to the diet is desirable. One teaspoonful of a one per cent solution of iron and ammonium citrate added to the feeding is usually a sufficient daily supply. Excessive amounts of iron salts are irritating to the gastrointestinal tract.

By the time prematurely born infants have attained the weight of six pounds (2700 grams) they may be treated in much the same manner as young full-term infants and the treatment thereafter will be essentially the same as that of average normal infants; the food requirements for a number of months, however, are likely to be relatively high in proportion to the body weight. Many infants who are free from disease will gain sufficiently rapidly to be of average size and weight by the age of six months. The majority, however, are somewhat undersized and underweight throughout the first year. Ultimately the development is as good as that of children who have been born at full term.

CHAPTER XXVIII

COMMON INFECTIONS WHICH ARE ASSOCIATED WITH NUTRITIONAL DISTURBANCES

An important reciprocal relationship exists between infections and nutrition in infancy. Infants whose diets have been inadequate in total fuel value or in such specific factors as certain of the vitamins or protein and who, as a result, have become undernourished, are especially susceptible to infections. On the other hand, any infection which gives rise to vomiting, diarrhea, cell destruction or increased metabolism, or which results in diminished secretion of the gastrointestinal juices or diminished absorption from the gastrointestinal tract necessarily affects nutrition. Infants in general are more susceptible to infections of the rhinopharynx, middle ear, and urinary tract than are older persons; these infections are especially likely to exert a deleterious effect on nutrition.

Because of the fact that many of the symptoms produced as the result of infections are similar to those resulting from an unsuitable diet, a frequent error is made in assuming that the food is at fault when the difficulty is really infection. On the mistaken assumption that the symptoms are due to unsuitability of the food, the diet often is changed by decreasing the amounts of various food components—a change which leads only to still greater impairment of the nutrition and to further susceptibility to infection. Certain infections in infancy are so common and so frequently associated with nutritional and gastrointestinal disturbances as to deserve special and somewhat detailed consideration.

Rhinopharyngitis

Rhinopharyngitis, or simple “cold in the head,” is of relatively frequent occurrence and of little importance in itself except in so far as extension of the infection to the

middle ear, bronchi, or lungs is concerned. Infants otherwise in good condition who develop acute rhinopharyngitis have slight fever, refuse a portion of the feedings, and occasionally vomit. The stools may be somewhat more numerous. The symptoms persist for only a day or two and require no modification of the character of the feeding, although the infant himself will limit the intake. It is doubtful whether the course of an acute rhinopharyngitis can be shortened materially by therapy, although local treatment may make the infant more comfortable and may prevent extension of the infection. In the early stage of rhinitis it is useful to keep the nose open by occasional use of $\frac{1}{2}$ to 1 per cent ephedrine in saline if it does not prove irritating. The amount used should be small, one drop on each side often being sufficient. After three or four days when the secretion has become purulent, suction is advisable to clear the nose. If silver protein is used, a 5 per cent solution is satisfactory. However, the usefulness of silver protein is somewhat questionable. Maintenance of increased environmental humidity is helpful, especially in preventing complications.

Otitis Media

Otitis media is one of the frequent complications of rhinopharyngitis, infants being especially susceptible to this infection because of the fact that the eustachian tubes are short and relatively wide and are more nearly horizontal when the infant is lying on his back. One of the first symptoms of otitis media in addition to restlessness and fretfulness is likely to be vomiting. Vomiting may occur even before fever or local evidence of inflammation on otologic examination. It may be persistent and projectile. Later, diarrhea may develop. The severity of the diarrhea depends on the nutritional condition of the infant and on the severity and virulence of the infection.

In well-nourished infants, an ordinary acute otitis media is not likely to be followed by very severe diarrhea, and the

diarrhea ceases rather promptly on suitable treatment of the ear infection. Occasionally middle ear infections occur in epidemic form; these usually are due to toxin-producing strains of the streptococcus. The diarrhea is then more severe and may lead to marked dehydration, with acidosis and severe toxemia, the clinical picture being that of "alimentary intoxication" (see p. 282).

Some degree of fever is present in any type of otitis media, although markedly malnourished infants may show little temperature reaction. The infant's actions may give no clue as to the location of the infection, although there may be some rolling of the head and occasional pulling at one ear in the case of older infants. The diagnosis can be made only by otologic examination. For this purpose the electric otoscope with magnifying glass attachment is especially useful, although with experience, one blessed with good eyesight may make a satisfactory examination with the head mirror and speculum. The infant's tympanic membrane is more horizontally placed than that of the adult and somewhat more difficult to observe.

The first change in the drum membrane in the presence of a developing otitis media is a congestion about the periphery and along the handle of the malleus. The membrane then becomes somewhat swollen and edematous and loses its luster. As serum and pus accumulate in the middle ear, some bulging occurs, and the color of the membrane, which has previously been drab, may become a fiery red. At this stage the landmarks are distorted and the light reflexes lost. Bulging may subside, due to discharge of pus through the eustachian tube, but the drum membrane remains red and thickened. In very undernourished infants and in those who are anemic or badly desiccated, the drum membrane may not be red even when pus is present in the middle ear. It may be a dull whitish color. After restoration of body fluids and transfusion, more active signs of inflammation are present.

The treatment to be instituted will depend on the severity and degree of extension of the infection and the state of nutrition of the infant. In healthy, well-nourished infants, middle ear infections often subside without paracentesis being performed, even though the tympanic membrane is red and moderately bulging. Recovery in these instances is presumably due to drainage through the eustachian tubes and overcoming of the infection by natural resistance. Local treatment of the nose as recommended for rhinopharyngitis is of some value. Ephedrine is especially useful in causing a shrinking of the edematous mucosa surrounding the openings of the eustachian tubes. The instillation of drops containing phenol and glycerin in the ear often relieves pain by the local anesthetic action of phenol, allows the child to get some much needed rest, and possibly has some effect in overcoming the infection, although one could hardly expect very much phenol to find its way through the eardrum. A 5 to 8 per cent solution of phenol in glycerin is recommended, and may be used, slightly warmed, as often as every half-hour. Certain proprietary remedies also are very useful.

Unless the temperature falls and evidences of infection subside within two or three days, paracentesis should be performed. Paracentesis is not often required in cases of otitis media occurring in well-nourished infants, but is required in almost all cases of otitis media in undernourished and athreptic infants. Paracentesis should be done at the point of greatest bulging of the drum. After paracentesis the ears should be kept clean, preferably by the use of dry cotton pledgets, though some physicians use gentle syringing with sterile physiological salt solution. Reincision may be required if the opening closes before subsidence of the infection. After adequate drainage of the tympanic cavity is established, cessation of the general symptoms may be expected, though the ears may drain for a considerable time, especially in undernourished infants. Profuse drainage continuing for longer than a week or two

may indicate poor resistance on the part of the infant, re-infection by way of the eustachian tubes, or extension of the infection to the mastoid antrum. A rare cause of continued discharge is tuberculosis of the middle ear.

In undernourished infants with poor resistance, the giving of repeated transfusions and the feeding of an adequate diet offer the best means of improving the resistance. Re-infection of the middle ear by way of the eustachian tubes is most often seen when an infected mass of adenoids is present in the rhinopharynx. Chronically infected tonsils may also be a factor in keeping up ear infection. The indication in these cases is removal of the adenoids or tonsils or both. When the infection has extended to the mastoid antrum, there may be definite local evidences of swelling and edema over the mastoid process or merely a bulging of the posterior superior canal wall and adjacent drum membrane. In some cases roentgenographic evidence of mastoid involvement is obtainable; however, many mastoid infections in infants are not revealed by the roentgenogram. When definite mastoid infection is present, drainage of the mastoid antrum or a more extensive mastoid operation is indicated.

The treatment of such gastrointestinal symptoms as may be present is that outlined in the chapters on Diarrhea and Vomiting. The infant should not be forced to take food beyond the desires of his appetite, and water should be supplied freely especially if any evidences of dehydration have developed. Often fluid must be supplied parenterally.

Mastoiditis

Probably in the majority of instances of otitis media, extension of the infection to the attic and mastoid antrum occurs, especially if much pus has accumulated. The mastoid antrum in the infant is present at birth as a single small cell. Later this cell enlarges and with growth and pneumatization of the mastoid processes communicates with other cells. During early infancy the antrum and

middle ear form essentially one cavity, communicating through the *aditus ad antrum*. When the tympanic cavity is drained, the antrum also drains unless obstruction occurs at the aditus. Such obstruction may occur, however, if the aditus is narrow, as the mucosa easily swells so as to shut off completely the antrum from the tympanic cavity. In these instances the infection remains in the antrum and may give rise to constitutional manifestations. In infants who are undernourished and whose resistance is lowered, infection in the antrum tends to spread into the adjacent bone, even though blocking off at the aditus may not be complete. It is in such infants that severe constitutional symptoms are seen and in whom the infectious process in the antrum persists despite free drainage of the middle ear through a tympanic opening.

A variety of organisms may be responsible for mastoid antrum infections, hemolytic streptococci being especially serious offenders. The general symptoms of mastoid antrum infection do not usually differ greatly from those of middle ear infections with the same organism except that the symptoms are likely to be somewhat more marked and to continue for longer periods of time. In many cases of mastoid infection with such organisms as staphylococci, in infants that are well nourished, no effects other than those of any febrile disturbance may be observed, even though large subperiosteal abscesses occur. Little or no vomiting or diarrhea and no tendency to anhydremia may be noted. On the other hand, in malnourished infants with hemolytic streptococcus infections, or with mastoid infection with organisms of the intestinal group, marked general symptoms of diarrhea, vomiting, and anhydremia may be present despite the fact that local evidences of mastoid infection are slight. When a baby is encountered who suddenly has developed the symptoms of cholera infantum, mastoiditis should be suspected and the diagnosis should be immediately confirmed or excluded. If mastoiditis is present as the cause of the symptoms, recovery from the serious

phase of the illness begins promptly when the affected mastoid antrum is drained. The mastoiditis more often is bilateral than unilateral. The diagnosis of mastoiditis as it occurs in relation to cholera infantum often requires an experienced otologist because of the dearth of local evidence of the disease.

The local evidences in some instances amount to no more than a swelling of the posterior superior canal wall and adjacent drum membrane. There may be no tenderness, redness, or swelling over the mastoid region. Roentgenograms may reveal a clouding of the antrum and even of the whole mastoid process; but inasmuch as there is so much individual variation in normal infants in the degree of pneumatization of the mastoid and in the size of the antrum, roentgenograms should be interpreted only in comparison with the opposite side, and if this also is involved, it may be impossible to reach a conclusion as to the significance of the radiographic findings. In some cases a definite diagnosis of mastoid or mastoid antrum involvement cannot be made. The condition can be suspected only on the basis of the symptoms and by exclusion of other causes for the fever or general symptoms. Not infrequently extensive mastoid involvement is discovered at autopsy when very little evidence of the condition had been present up to the time of death. Occasionally otitis media may subside entirely, but at autopsy the mastoid antrum is found filled with pus. When mastoid infections of the type described are recognized and suitably treated by surgical drainage, prompt disappearance of the general symptoms follows in the majority of instances.

Another type of mastoid involvement is seen almost exclusively in malnourished infants and appears to be secondary to gastrointestinal disturbance or to intestinal infection. In these infants the middle ear and mastoid involvement is not present until late in the course of diarrhea. The ear infection is not preceded by rhinopharyngitis. The local evidences in the tympanic membrane are slighter

than in the case of the primary ear infections and the local evidences of the mastoid involvement are likewise slight, even in cases in which an extensive necrosis is found at autopsy. In these secondary types of mastoid involvement, the organisms found are, in most instances, intestinal types, such as strains of the *E. coli*, or members of the paratyphoid or paradysentery groups. The organisms obtained on culture from the mastoid antrum are often different from those obtained from the middle ear, thus suggesting the possibility of direct blood-stream infection, although it appears probable in most instances that the intestinal organisms reach the ears from vomited material by way of the eustachian tubes.

Coincident with the development of mastoid infections in infants suffering from diarrhea, usually an exacerbation in the gastrointestinal symptoms and an elevation of the temperature above the previous level will be observed. In these cases, drainage of the middle ear and mastoid antrum does not result in the same degree of improvement in the general and gastrointestinal symptoms as is observed in primary types of ear infection previously described. The mortality in these cases is high because of the fact that the infants who develop the complication are already in a precarious condition and are still further weakened by the added infection.

Still another type of mastoid infection occurs occasionally in infants. It is characterized in part by low-grade chronicity and an almost complete lack of the symptoms and signs ordinarily attributed to mastoiditis. The otologic signs are no more than a moderate edema or bulging of the posterior superior canal wall, changes that are overlooked easily by the inexperienced. The most constant symptoms are occasional refusal of part of the food, occasional and moderate regurgitation, and the passage of stools with only slightly greater than normal frequency. The stools are sufficiently acid to excoriate the skin of the anal region, and no customary diet change decreases the stool acidity ap-

preciably. When sufficient food is given, these babies make excellent growth progress and exhibit no evidence of illness other than the mild gastrointestinal symptoms that have been mentioned. The body temperature is normal. This variety of mastoiditis is important from two major standpoints. The presence of gastrointestinal symptoms, even though moderate, too often leads to therapeutic underfeeding in the attempt to correct the digestive disturbance. Further reductions in diet are made because the first ones did not cause improvement. Marked malnutrition inevitably results from such a regimen. The other important effect of this type of mastoiditis is the possible occurrence of attacks of syncope, which may result in sudden and unexpected death. If the infant is observed at the time of occurrence of a syncopal attack, he may be revived easily by suitable stimulation. In these circumstances the attack is of only brief duration and, when it is over, the baby seems none the worse and in his usual state of health and vigor. In many instances, no doubt, death from so-called status lymphaticus in infancy is caused by chronic low-grade, undiagnosed mastoiditis. Fortunately syncopal attacks and death from this cause are not common.

The treatment of mastoid infections in infants depends on the condition of the infant and the severity of the process. In many instances the mastoid infection in well-nourished infants subsides spontaneously or after adequate middle ear drainage. In others, it becomes necessary to drain the mastoid antrum. In cases of antrum infection without extension to the remainder of the mastoid process, antrotomy or "post-auricular drainage" is all that is required. This simple operation is performed under local anesthesia and may be accomplished with very little disturbance or shock. The operation consists merely in the removal of a small button of bone over the mastoid antrum. The pus in the antrum should be gently removed with a swab, and a gauze drain should be inserted. Extensive curettage should not be attempted, especially in the case

of malnourished infants, as this may damage the mucosa and adjacent bone and favor extension of the process. The wound should be kept open for a reasonable time by the insertion of a drain. Healing is usually prompt in well-nourished infants, but in athreptic infants is, at times, very slow, there being often but little evidence of repair for a period of weeks. In cases of extensive involvement of the mastoid process, more radical operation is required. In all cases of mastoid infection, suitable treatment of the rhinopharynx and middle ear should be continued and the fluid balance of the body should be maintained, using parenteral routes of administration if necessary.

Sinusitis

The paranasal sinuses are only partially developed at birth. However, even at this early age, the maxillary and ethmoidal sinuses are sufficiently developed to be of clinical importance. These sinuses are involved with every generalized infection of the nasal mucosa. Usually, when babies have colds, the sinus infection is of no great clinical importance because it subsides spontaneously without special treatment. In occasional instances, especially in the case of maxillary sinusitis, drainage from the sinus is blocked by swelling of the mucous membrane. Blockage of drainage in the case of purulent sinusitis produces marked symptoms at any age. In infancy these symptoms are likely to include those which characterize cholera infantum or alimentary intoxication. Thus sinusitis may have a causal relationship to one of the most serious illnesses of infancy. Fortunately this event is not common, but when it occurs, its recognition is extremely important. In the diagnosis roentgenograms are helpful. Roentgenograms may or may not give a true picture of the status of the inflammation of the sinuses, but they show the location and stage of development, particularly of the maxillary antra; this information is of assistance if diagnostic puncture of the antra is to be done. When cholera infantum

is dependent on sinusitis, recovery occurs as a result of local treatment. The recovery is not quite so prompt and dramatic as that which occurs after mastoidectomy in those instances in which the illness is dependent on mastoiditis. With the institution of local treatment to the sinuses, the progression of the constitutional illness is arrested, and within two or three days rapid improvement begins. The difference in the course of these two conditions probably is dependent on the difference in adequacy of drainage.

Pyelitis

Pyelitis is a frequent cause of nutritional disturbance during infancy and one which is likely to be overlooked, since no symptoms directly referable to the urinary tract may be observed. The frequent occurrence of pyelitis in infants may be explained on the basis of lack of immunity to colon bacillus infections, to anatomical peculiarities of the urinary tract and to frequent soiling about the urethra. Pyelitis may occur in well-nourished infants but is distinctly more frequent in those whose nutrition is below normal and in those whose resistance is lowered by the presence of other infections. Thus the development of pyelitis is often observed after acute or chronic rhinopharyngeal or ear infections. The first evidences of pyelitis may appear after an infant has suffered from a gastrointestinal disturbance. It is possible that the intestinal wall becomes permeable to colon bacilli in such circumstances, and that the organisms reach the kidney by way of the blood stream or lymphatics. The development of pyelitis is favored by any anomaly of the urinary tract, such as strictures or kinks in the ureters or disturbances in the neuromuscular function of the bladder. Pyelitis is much more frequent in females than in males.

Whatever the cause of pyelitis, the effects on nutrition may be marked. The infant's appetite is lessened and vomiting is of frequent occurrence. There is occasionally diarrhea. Even infants who take fair amounts of food and

do not have much gastrointestinal disturbance fail to show normal gain in weight. Infants with pyelitis may be pale and pasty in appearance and are often languid and apathetic.

A definite diagnosis of pyelitis can be made only by urinary examination. A voided specimen of urine is satisfactory for examination in the case of males, but in females a catheterized specimen is preferable because of the possibility of contamination with pus cells from the vagina or labia. The methods of collecting urine are described in Chapter XXXII. An uncentrifuged specimen should be examined under the high power of the microscope. The diagnosis of pyelitis is warranted only if there are more than 8 or 10 leucocytes to a field, especially if they are in clumps. A single urine specimen may be free from pus and subsequent specimens may contain large amounts. This finding is probably due to retention at some point in the kidney or ureter.

In the treatment of pyelitis many remedies have been used, each having been supplanted by one more recently discovered. At present, sulfonamide therapy is the procedure of choice. Effective concentration in the urine may be obtained with one-half the dosage customary for other infections. For most of the organisms causing pyuria a concentration of 10 mg. for each 100 ml. of urine is effective. A few organisms require somewhat more. An effective concentration in the urine is attained with a twenty-four-hour dosage of 0.1 Gm. for each kilogram ($\frac{3}{4}$ gr. for each pound). In case of sulfonamide sensitivity or resistance some of the older remedies can be used and are effective. These include mandelic acid and hexamethylenamine.

Hexamethylenamine (methenamine) is administered by mouth. This substance is excreted in the urine and, in the presence of acid in the urine, it is partially decomposed, with the liberation of formaldehyde. The dosage of methenamine for an infant is from 0.2 Gm. to 0.5 Gm.

(3 to 7.5 gr.) as often as four times a day. Coincident with the administration of methenamine, substances should be administered which will render the urine acid in reaction in order that the methenamine may be effective. The most suitable substance to use for this latter purpose is ammonium chloride, which may be given in a dosage about equaling that of the methenamine. Methenamine administration, especially in the acute stages of pyelitis, may cause temporary hematuria. In case hematuria develops, the drug administration should be discontinued. It may be necessary to continue methenamine treatment for long periods before the urine becomes free from pus. During the period of methenamine administration, fluids should be restricted. In the presence of anhydremia with acidosis, methenamine administration should not be attempted as acid should not be administered nor should fluids be restricted.

By means of these various methods of treatment the urine can be made sterile and free from pus in nearly all instances. In some cases pyuria recurs, sometimes repeatedly. Recurrences may be associated with infections elsewhere in the body, as in the adenoids and tonsils. When such infections are present, they should be treated appropriately. Recurrences also may be associated with obstruction, usually from anatomical anomalies. In all instances of recurring pyuria special studies should be made of the urinary tract. Urograms may be made after intravenous administration of certain substances which are excreted in concentration by the kidneys and which are opaque to x-rays. If this procedure gives unsatisfactory results, retrograde pyelograms are indicated. After cystoscopy, opaque material is passed into the kidney pelvis by way of a ureteral catheter. It is important that obstruction be relieved and anomalies be corrected in so far as is possible.

Tuberculosis

Two possible sources of tuberculosis commonly exist for the infant. One source is the human beings in his immediate environment, and the other is the cow's milk with which he is fed. In this country, tuberculosis acquired from the milk supply is relatively infrequent, because of the exclusion of tuberculous cattle from dairy herds and because of pasteurization of the milk supply. If the milk for infant feeding is boiled, as is recommended, transmission of tuberculosis by way of the milk becomes impossible.

Regardless of the incidence of tuberculosis among the population of any community, it is high in any family in which the disease exists in an active stage in any member. The infant needs protection from this source, chiefly. An infant born to a tuberculous mother should not be permitted to have contact with the mother at any time after birth. If tuberculosis exists in a family, some means must be devised to prevent contact of the baby with the infected member. If the social and economic circumstances are such that the baby cannot be cared for away from the infected person, efforts should be made to teach the infected member how to avoid the spread of his infection. Prevention is by far the most important aspect of the control of tuberculosis.

Should the baby acquire tuberculosis, the infection may or may not affect his nutrition adversely. The effect on his nutrition and on his general health depends largely on the extent and severity of his infection. A strong tendency to recovery from the first infection in tuberculosis exists. Recovery is more difficult in instances of massive infection. Massive infection is more likely with continued or repeated exposure to some member of the family group with active disease. Removal of the baby from a tuberculous environment is the first step not only in the prevention of this disease, but also in its treatment.

It is not appropriate to think of all babies with tuberculosis as being malnourished. The infection may exist without any discernible effect on the health and well-being of the infant. In these instances the disease may be detected often only by a most careful examination. No nutritional precautions need be taken other than should be taken for all babies. The diet should be ample and complete.

With massive infection and extensive disease, malnutrition is an expected result. In these circumstances malnutrition may persist until the infant starts to recover, despite all efforts to improve the nutritional status. No attempt at stuffing with food is indicated; instead, the prescribed diet should be much the same as would be given during health. Some loss of appetite may interfere with ingestion of part of the food offered. Gastrointestinal symptoms, such as vomiting and diarrhea, are not ordinarily a part of the illness.

Syphilis

Congenital syphilis has been reputed to be a cause of marked malnutrition. However, malnutrition occurs much less frequently in association with infantile syphilis than was formerly the case. This change in relationship is due in part to improvements in artificial feeding and in part to earlier recognition, by means of serological tests, of the presence of infection in the baby, and to more efficient antisyphilitic therapy.

All but a very few syphilitic babies are free from external evidence of their infection at birth and in the first few weeks after birth. During this period the nutrition usually is unimpaired and the babies appear entirely normal. If antisyphilitic treatment is begun as soon as the diagnosis of syphilis can be made, and if the treatment is carried out properly and effectively, no impairment of nutrition is to be expected and growth will proceed in a normal manner.

On the other hand, if the presence of the infection has been overlooked and if at the same time the infection is relatively severe, the nutrition is likely to suffer until such time as the activity of the infection can be brought under control by treatment. Mild infections, even if neglected for a time, usually do not impair the nutrition greatly. Even when the infection is extensive, the health and nutrition are affected only until the routine course of therapy is well established and external evidence of the disease is no longer manifest. The critical period for the syphilitic infant is during the first few weeks of manifest disease and during the period when therapy is being started. It is during this period that death of the infant is most likely to occur. Mortality among syphilitic babies still is high, usually approximating 20 per cent. The mortality would be low, were all syphilitic babies treated at the earliest period that recognition of the presence of the infection is possible. It is chiefly in instances of neglected infections, and in those diagnosed after much damage has been done, that deaths occur.

When the infection is severe and extensive, death may occur as a direct result of the damage caused by the infection. By means of modern antisymphilitic remedies a large proportion of the spirochetes may be destroyed promptly, but, unfortunately, if penicillin or one of the arsenical antisymphilitic drugs is given in anything like a full dose to a baby with extensive syphilitic infection, the baby is likely to die shortly afterward as a direct result of the treatment. Consequently, in the treatment of severely infected babies a middle course must be steered between death by the disease and death by treatment. Any penicillin or arsenical medication should be preceded by treatment with mercury or bismuth. Mercury is somewhat preferable because it is less spirocheticidal than bismuth, and damage cannot be done by customary therapeutic dosage. Gray powder (mercury with chalk) is a useful preparation and can be given in a dosage of one-fifth or one-fourth grain (12-15

mg.) twice daily, increased to three times daily. If bismuth is chosen as the first drug to use, it can be given as the salicylate in oil intramuscularly at three-day intervals in a dose of 2 mg. for each kilogram. After a week or ten days of treatment with mercury or bismuth, regression can be observed in the visible lesions, and the chosen arsenical drug can then be given tentatively in a fractional dose; the dose should not be more than one-fourth the full calculated dose. A second dose may be given two or three days after the first, in the amount of one-half the full dose. The dosage is gradually increased in this manner until the full amount is given at an injection. Treatment with mercury or bismuth is continued throughout the first period of arsenical treatment. By the time full-sized doses of the arsenical preparation are being given, the baby is ready to be established on whatever antisyphilitic treatment regimen may be chosen. Penicillin is a most useful remedy. If it is to be used, it should be given concomitantly with the arsenical preparation after the preliminary course of arsenic and heavy metal. Penicillin treatment is not yet sufficiently satisfactory to warrant its use alone, because of relapses in a considerable proportion of cases.

During the critical period of treatment of the syphilitic infant the state of nutrition is an important index of progress. If the infection is not being brought under control satisfactorily, or if the treatment is too intensive, the baby is likely to lose weight. Continued gain in weight during this period is an excellent criterion of progress. In order that the baby may gain satisfactorily, it is important that the feeding as well as the antisyphilitic treatment be managed properly. Because this critical period occurs chiefly in the first three months, it is usually possible for the mother to feed the baby at the breast. Human milk, if it is obtainable, is highly desirable during this period.

After babies have been established successfully on a regimen of antisyphilitic treatment, no reason ordinarily exists why they should not make the same nutritional and

growth progress as normal nonsyphilitic infants. The nutritional requirements are the same in the two instances, and the infection is held to a wholly inactive state by the continued therapy.

No contraindication exists to the feeding of a congenitally syphilitic infant at the breast of its own mother. The mother also is syphilitic, even though she shows no evidence of the disease; otherwise the infection could not have been transmitted to the infant. The mother cannot be reinfected by her infant. In the rare instances in which the baby acquires syphilis after birth and in which the mother has not been the source of the baby's infection, the infection is communicable to the mother through suckling the breast.

From the preceding discussion it is apparent that early diagnosis of syphilis in the infant is important. Serological tests with the blood of the mother, before or at the time of parturition, are increasingly customary. If the mother is shown by this means to have syphilis, the possibility of syphilis in the baby exists. Not all syphilitic mothers transmit the infection to their infants. The withholding of treatment is desirable in the case of an infant born to a syphilitic mother, until the diagnosis of syphilis can be established definitely in the baby. Close clinical and serological observation of the suspected baby is necessary during the first two months, or until the diagnosis of syphilis is made or excluded.

CHAPTER XXIX

RICKETS

Rickets is a disorder of nutrition characterized by impairment of utilization of calcium and phosphorus, of which the outstanding manifestation is poor mineralization of bone, with defective bone growth and often with abnormal changes in the shape of the bones. Changes of lesser importance occur also in the body musculature. Though impairment of calcium and phosphorus utilization may have several causes, the factor responsible almost exclusively for the production of rickets in the full-term infant is deficiency of vitamin D, or of ultraviolet energy that produces vitamin D in the body by the activation of 7-dehydrocholesterol in the skin. In the case of infants born prematurely, however, deficiency of calcium and phosphorus is important also as a cause of rickets.

Etiology

Vitamin D is present in the diet of the infant in only negligible amounts unless special thought is given to its inclusion. In those instances in which it is not added to the diet intentionally, the infant is dependent for protection against rickets chiefly on exposure to sunshine. For this reason rickets is a disease chiefly of the temperate zones, where climatic conditions prevent exposure to sunshine for long periods. The incidence of rickets is seasonal, the greatest frequency being observed in the spring, usually in March in the north temperate zone. Rickets is unusual in the tropics and occurs there only in instances in which the baby is kept closely housed. In the temperate zone the disease is observed more frequently in dark-skinned races. It occurs uncommonly among these races in their original native habitat because of the relatively large amount of

sunshine available; it becomes common when these peoples have migrated to regions with colder winters where less exposure to sunshine prevails. In these races the pigment of the skin obstructs the passage of the ultraviolet rays and a more intense exposure is necessary than for those without pigmented skin. In the frigid zones the customary diet of the natives includes fish and animal fats that supply adequate vitamin D. In these regions also, babies are more likely to be breast-fed, a procedure offering a considerable degree of protection against rickets.

Ultraviolet "light" has wave lengths shorter than does visible light. The zone in the ultraviolet spectrum that is chiefly effective against rickets is that portion with wave lengths from 230 to 310 millimicrons. Sunshine as it reaches the earth's surface under most favorable conditions contains waves usually no shorter than 290 millimicrons. The most favorable conditions exist when the sun is overhead and the air is free from fog, dust and clouds. The shorter the wave length, the more certainly are the ultraviolet rays filtered out by the air and the material floating in it. The greater the inclination of the sun, as at rising or setting, the less do the ultraviolet rays of the shorter wave lengths reach the earth. "Skyshine," or the light reflected from the sky, clouds or other light surfaces, is from one-half to two-thirds as effective in rickets prevention as direct sunshine. Thus it is apparent that sunshine is not effective as an antirachitic agent at all times and that the range of effective wave lengths reaching the earth from the sun is at best a narrow one. Furthermore, the usual window glass does not permit waves as short as 310 millimicrons to pass. Transparent materials that permit passage of ultraviolet rays have been devised to replace window glass, but in general the benefits derived are not commensurate with the expense. Ultraviolet rays with wave lengths shorter than those received from the sun are available only from artificial sources, such as a carbon arc light or a mercury vapor quartz lamp.

Babies subsisting on human milk have rickets less frequently than those artificially fed. The difference between these two groups is not one of difference in the amounts of vitamin D present naturally in the milks, but probably chiefly one of the ratio of calcium to phosphorus in the two milks, the ratio being more favorable for utilization in human milk. Perhaps also the more acid reaction of the content of the lower intestinal tract of the breast-fed baby is an aid to absorption of calcium and phosphorus. Even though this difference exists between the results of these two types of food, it is only one of degree. The breast-fed baby is not as efficient in utilizing calcium and phosphorus without vitamin D as with it, and severe rickets sometimes is observed in breast-fed infants.

For artificially fed babies the development of rickets or its prevention has no relationship to the kind of processing the milk may have had. Rickets is observed as frequently in infants fed raw milk as in those fed pasteurized, boiled, evaporated, or dried milk. Before vitamin D was given customarily to well infants, rickets was observed more frequently in babies fed sweetened condensed milk than when better proportioned formulas were given. Such an effect presumably was dependent on the smaller milk intake customary with this type of feeding.

Other dietary factors in the production of rickets have been mentioned in the literature, but they seem of little practical importance for the infant. Although rickets may be produced experimentally in animals by feeding diets deficient in either calcium or phosphorus, mineral deficiency in the diet is rarely the cause of infantile rickets, except possibly at times in the case of prematurely born infants. Human milk, when taken by the full-term baby in sufficient amounts to supply the fuel needs, also provides sufficient calcium and phosphorus. Cow's milk contains from three to four times as much calcium and from seven to eight times as much phosphorus as human milk, and even when diluted provides an abundance of these elements.

In certain restricted circumstances cereals have a rachitogenic effect. This effect is of no importance in infant feeding, but it may become important in a child population fed large amounts of porridge and insufficient amounts of milk. It is important also in assays of vitamin D in which the rat is the test animal. The rachitogenic effect of cereals is due to their content of phytic acid (inositol hexaphosphoric acid) or its sodium salt.

The diet most commonly used for producing rickets in rats for assay purposes is one relatively high in calcium and low in phosphorus. Much of the phosphorus of the diet is present as phytate. The phosphorus of phytate is not available to the rat without vitamin D and rats develop rickets because of inadequate phosphorus. This type of rachitogenic effect is not possible in an infant receiving a customary diet because of the relatively large amount of phosphorus easily available from the milk. He does not depend on cereal for phosphorus.

Phytic acid combines with calcium to form an insoluble and unutilizable compound. Thus the phytic acid of cereals makes unavailable for absorption some of the calcium of the diet. If a large amount of cereal and only a small amount of milk were fed, rickets might result from inadequate calcium. Cereals in the form of bread are less rachitogenic than cereals as porridge. During the rising of bread dough the enzyme phytase both of wheat and of yeast hydrolizes most of the phytate and destroys it. In the preparation of porridge, heat destroys phytase promptly and the phytate content remains high. If the amount of milk served with the porridge is ample to provide for neutralization of phytic acid and in addition to permit sufficient absorption of calcium, no rachitogenic effect can occur. The relative amounts of milk and cereal in customary diets for infants are such that no rachitogenic effect is to be expected.

In general, the diet of the mother during pregnancy should have little relationship to the development of rickets

in the infant, though it may have an effect in those instances in which the fault in the mother's diet is extreme. The fetus tends to get its share of calcium, even though it does so at the expense of the mother's stores. A few instances of congenital rickets have been reported, but these have been of babies whose mothers had osteomalacia. In any usual circumstances a baby well fed after birth and receiving vitamin D from an early age will not develop rickets, regardless of the mother's diet during pregnancy.

The changes characteristic of rickets cannot occur unless growth is proceeding actively. Also, the more rapid the growth of bone, the more easily do rachitic changes take place. When calcium and phosphorus utilization is altered in the adult in a manner similar to that which produces rickets in the infant, the disease resulting is known as osteomalacia. Rickets does not occur in babies who are malnourished to the extent that the growth rate is retarded. Unless precautions are taken by administering adequate vitamin D, rickets may occur as a result of any procedure that causes a rapid increase in growth. Thus, rapid recovery from malnutrition may be associated with rickets. The administration of thyroid to an infant whose growth has been retarded as a result of hypothyroidism is likely to cause a rapid increase in growth with attendant rachitic changes. A baby born prematurely is more susceptible to rickets than a baby born at term for two reasons. The growth of the prematurely born baby is relatively more rapid; also he is handicapped by a relatively great demand for calcium and phosphorus for the formation of new bone and at the same time by a limited capacity for ingesting that which is needed. Twin babies often are affected in the same manner as are babies born prematurely, since usually they are smaller than the average baby born at term and are expected to grow more rapidly.

Since rate of growth is an important factor in the production of rickets, this disease occurs as a result of vitamin D deficiency only at those ages at which growth is most

rapid. Rickets of noteworthy degree seldom develops before six months of age, even though growth is rapid. It is observed with greatest frequency between the ages of 6 and 18 months, after which time the incidence declines. Another period of rapid growth occurs at the time of puberty. Rickets at this age probably is nonexistent in the United States, but it has been reported for girls in India who have been kept closely housed because of the purdah system.

The frequency of rickets among babies is difficult to determine with accuracy. Prior to the time (*circa* 1920) when the administration of vitamin D to babies began to be routine as a preventive measure, the prevalence of rickets was marked. The knowledge that vitamin D is necessary for the infant gradually spread until now few mothers and no physicians are unaware of this need, and in a high proportion of instances something has been done about satisfying it. Rickets still is found with some frequency in some of our larger cities, especially among the dark-skinned races, but it has almost disappeared in the country as a whole. At many medical schools the finding of babies with rickets to use as examples in teaching has become difficult. Estimates of incidence as they appear in the literature must be accepted with caution because of the extreme delicacy of the criteria sometimes used in making diagnostic surveys. An estimate made in New York City early in the 1930's indicated that approximately 15 per cent of babies chosen at random from a low economic level had rickets of a clinically significant degree.

In this country rickets from deficiency of vitamin D occurs only in the first two or three years of life. Rickets from other causes occurs chiefly after this age period. These two groups sometimes are distinguished by the designations infantile rickets and late rickets. It is rare to find the late type of rickets occurring in infancy. Rickets at any age is caused by a disturbance of calcium and phosphorus metabolism. In late rickets this disturbance is most

often brought about by a deficiency of utilization of vitamin D or by an altered acid-base equilibrium with the production of chronic low-grade acidosis. Rickets caused by deficiency of utilization of absorbed vitamin D is often called "resistant rickets." The clinical and blood pictures are similar to those of infantile rickets except that excessive amounts of vitamin D, often several hundred thousand units daily, are needed for cure. A child with chronic low-grade acidosis often develops rickets. The acidosis interferes with the deposition of mineral in bone. The bone changes, as seen radiographically, are identical with those of infantile rickets produced by vitamin D deficiency. Any one of several causes may produce chronic acidosis, but the common one is kidney dysfunction of a mild type not easily detectable by the usual routine examination. When more obvious chronic nephritis is present, phosphate retention may become an additional factor in interfering with calcium deposition in bone. This condition of "renal rickets" is observed chiefly during middle childhood, but it may occur in late infancy when congenital malformations of the kidney are present. Children with uncontrolled celiac disease occasionally have the bone changes of rickets; in this condition the failure of mineralization of bone presumably is dependent on impairment of absorption from the intestinal tract.

Rickets is not ordinarily associated with disturbances of any of the endocrine glands. Changes indistinguishable from rickets occasionally are observed in association with dysfunction of the parathyroid glands. Parathyroid insufficiency leads to a low calcium content of the blood, to tetany and to defective bone mineralization.

Pathogenesis and Pathology

The most characteristic manifestation of rickets is failure of calcification of the growing portion of bones, a disturbance most easily demonstrated radiographically in the metaphyses of the long bones. Under normal conditions

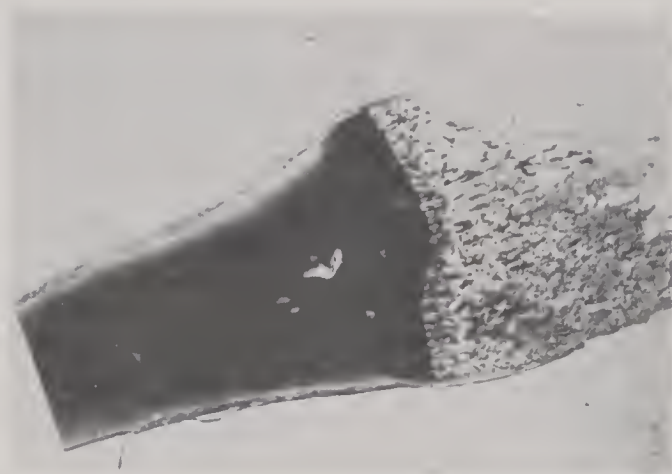
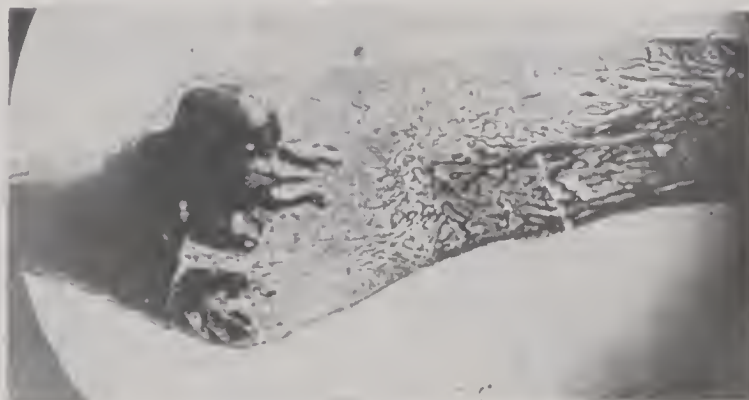
*A.**B.**C.*

Fig. 12.—Sections of normal (*A*) and rachitic (*B* and *C*) ribs. Note regular line of calcification at the costochondral junction in the normal rib and the irregular line in the rachitic ribs. The softened rachitic ribs show bending inward as the result of external air pressure.

of growth the long bones increase in length by the production of an organic matrix or osteoid tissue. This matrix is promptly filled with a deposit consisting chiefly of calcium phosphate with small amounts of calcium carbonate; the calcification occurs so promptly that at any one time only a thin layer of uncalcified osteoid tissue is to be found. As growth proceeds further, that part of the more or less solidly calcified osteoid tissue lying nearest the shaft of the bone becomes transformed into cancellous bone in the interior and normal supporting bone on the exterior. With the failure of calcification characteristic of rickets the newly formed osteoid tissue becomes mineralized inadequately and irregularly. When the rickets has been of considerable duration, the amount of uncalcified osteoid substance is relatively large; having no solid support it tends to "mushroom," causing enlargement at the ends of the bones. The enlargement often is such that it is easily detectable on inspection and palpation. The mushrooming of the osteoid tissue usually is not apparent in the roentgenogram because little or no shadow is cast by this organic tissue. Radiographs show the irregularity of calcification and the flaring or widening of the end of the calcified portion of the bone; usually also "cupping" is manifest, produced by a lessened amount of calcification at the center as compared with the periphery. (Figs. 12, 13, 14, 15.) Though the growing portion of bone is affected in the most striking manner, the remainder of the bone also shows the effect of insufficient available mineral; rarefaction may be observed when the radiograph is compared with one of normal bone. Such bones are easily deformed by the stresses of weight-bearing or even by the force exerted by the musculature. Fractures may occur (Fig. 16). Centers of ossification that should make their appearance in the roentgenogram do not become mineralized and their radiographic appearance is delayed. This phenomenon is most easily observed in the epiphyses and in the carpal bones. (Figs. 13, 15.)



Figs. 13.—Rickets. Irregular line of calcification at lower end of radius, with cuplike contour.



Fig. 14.—Rickets. Irregular calcification at ends of long bones with distortion.
Fractures of tibia and fibula.



Fig. 15.—Severe rickets. Rarefaction of bones. Incomplete ossification of epiphyseal centers.

The failure to deposit calcium salts in the bones is not attributable to lack of calcium in the circulating blood, for except in certain rare forms of rickets the blood calcium content is within the normal range, though usually at the lower limit of normal. On the other hand, the phosphate content of the serum is markedly diminished. The normal amount of inorganic phosphate in the serum of infants, expressed in terms of phosphorus, is from 5 to 7 mg. for each 100 ml. In active rickets the amount of phosphorus may be as low as 1 or 2 mg. for each 100 ml. of serum. The ester phosphorus of red blood cells is also markedly lowered in active rickets. The low phosphate content of the blood is not the result of deficiency of phosphorus in the diet, nor may the amount be raised appreciably by the administration of extra phosphate. The administration of vitamin D or the exposure of the body to sunshine or to artificial sources of ultraviolet rays is followed by a rise of the corpuscle ester phosphorus and of the inorganic phosphorus of the blood plasma, and by healing of the rachitic process. When the healing process is continuous, no marked change occurs in the calcium content of the blood, but retention of larger amounts of calcium from the food occurs. The increased amount of calcium and most of the increased amount of phosphorus retained from the food are deposited in the bones.

Considering 9 mg. of calcium and 5 mg. of phosphorus for each 100 ml. of serum as the lowest limit of normal values, and multiplying one of these values by the other, the resulting product is 45. The observation has been made that when this product is more than 40, rickets either is not present or is healing. Products below 30 are indicative of active rickets.

Phosphatase is an enzyme that exists normally in the blood. It is measured in terms of arbitrary units. The amount of phosphatase is increased in rickets, sometimes to a marked degree. Phosphatase determinations are of considerable assistance in estimating the relative activity

of rickets. Values of less than 15 Bodansky units for each 100 ml. of serum indicate that active rickets is not present. When rickets is active, values as high as 60 units or more may be observed.

Although the most easily demonstrable changes in rickets occur in the bones, rickets is not merely a disease of the bones but a general disease affecting other parts of the body as well. The muscles throughout the body are soft, flabby, and atonic, and may show slight microscopic changes at necropsy. The smooth musculature of the intestinal tract also is hypotonic. The ligaments are lax. A catarrhal condition of the mucous membranes of the respiratory and gastrointestinal tracts is commonly observed, but it is not certain that this condition depends primarily on the presence of rickets. In a fair proportion of cases of rickets the spleen is palpable; the enlargement may be extreme when a severe degree of anemia is associated with the rickets. The changes in the spleen are those of simple hyperplasia. The tonsils, adenoids, and cervical lymph glands frequently are enlarged, but this enlargement probably is due not so much to rickets as to the chronic infections to which rachitic infants are susceptible. The bone marrow often shows some degree of atrophy, with a deficiency of the cellular components. Secondary anemia usually accompanies the bone marrow changes. Whether the bone marrow changes and the anemia are definitely a part of rickets is not clear; it is possible that coexisting deficiencies, such as of iron, are responsible at least in part. In some cases the anemia is severe and is associated with lymphocytosis and marked splenic enlargement and corresponds closely to the von Jaksch's type of pseudoleukemic anemia or splenic anemia.

Symptoms

The most characteristic symptoms of rickets are referable to the bone changes. The bones throughout the body are not always affected to an equal degree: in some in-

infants the most marked changes are in the cranium, in others, in the extremities or thorax.

In cases of fairly marked rickets the head appears large, due chiefly to the development of thickened areas or bosses in the temporal and parietal regions (Fig. 16). The top of the skull is flat and often depressed toward the middle.



Fig. 16.—Rickets. Square head, frontal bosses and enlarged costochondral junctions ("rachitic rosary").

The anterior fontanel does not close at the regular time and may be widely open, even after the age of two years. Areas of softening of the cranial bones (*craniotabes*) may be detected on palpation. These areas are most frequently found in the neighborhood of the lambdoidal sutures either

in the parietal or occipital bones. Such soft areas may be present in the skulls of newly born infants who show no other manifestations of rickets; hence, craniotabes is not of significance unless developed during infancy.

In the thorax, the costochondral junctions are enlarged and may be felt as rows of beads running parallel to the sternum and curving outward toward the lower end of the chest (the *rachitic rosary*) (Fig. 16). In cases of marked and long-continued rickets, deformity of the chest occurs, with sinking in along the lines of the costochondral junctions. (Figs. 16, 17, 18.) In these cases the bending in of the ribs and cartilages may be sufficient to interfere with normal expansion. Associated with the sinking in of the thorax is a flaring of the lower ribs covering the liver and upper portions of the abdomen. At the upper edge of the flaring following the line of the diaphragmatic attachment a gutter-like depression occurs (Harrison's groove). The lower end of the sternum may be retracted so as to give rise to a funnel-shaped depression; such a deformity, however, may be congenital or the result of other causes, as, for example, respiratory obstruction due to adenoids.

The long bones of the extremities may show more or less deformity, depending on the degree and duration of rickets and the amount of mechanical stress to which the bones have been subjected. Some enlargement of the metaphyses occurs at the wrists in almost all cases of rickets. In children who are old enough to sit up or to stand, bowing of the thighs and legs occurs (Figs. 17 and 19). The rachitic child tends to sit in a cross-legged, tailor-like fashion, resting a part of the weight upon the arms; this causes deformity of both arms and legs (Fig. 18). Instead of being symmetrically bowed, the legs may be twisted in almost any direction (Figs. 19, 20, and 21). Although knock-knee occurs as a result of rickets, it occurs also from other causes. The most frequent cause of



Fig. 17.—Rickets. Deformities of head, chest and extremities.



Fig. 18.—Rickets. Characteristic posture, deformities of chest and extremities, "potbelly."

moderate knock-knee is the stress put on the knees by standing on flat, pronated feet.

Because of the lax musculature, the rachitic infant tends to slump into a heap when an effort is made to sit up. Ultimately this may lead to a considerable degree of kyphosis.



Fig. 19.—Rickets. Deformities of extremities, bowlegs and knock-knee.

The laxity of the muscles and ligaments of the feet predisposes to the development of flat feet. The lax musculature of the abdomen and of the intestines causes abdominal protuberance or "pot-belly" (Fig. 18). Intestinal atony results in constipation. Diastasis of the rectus abdominis muscles and umbilical hernia are common. As a result of muscular weakness, sitting, standing and walking are



Fig. 20.—Rickets. Rarefaction and deformities of bones. Knock-knee.

delayed, or, if the baby has learned to perform these acts previously, they may cease during the period of activity of the rickets.

Dentition is delayed, but the temporary teeth are not necessarily abnormal in shape or structure, since these teeth are fairly well developed at the time of birth (even though unerupted). Rickets during infancy is more likely to cause an alteration in the structure of the permanent teeth which are developing during the time that rickets is active.



Fig. 21.—Rickets. End result of severe rickets during infancy.

The rachitic child may be fretful, and irritable, may sleep poorly, and be restless during sleep. Excessive sweating about the head is common, although it is not definitely a manifestation of rickets. The infant may be pale and pasty in appearance. Rachitic infants are somewhat more subject to infections, particularly of the respiratory tract, than are normal infants. The chief reason for increased susceptibility appears to be the deficiency of vitamin D. Deficiency of any of the nutritional essentials leads to decrease in resistance to infection, and vitamin D deficiency is no exception.

Tetany may be associated with rickets, and when present there are the added symptoms of neuromuscular irritability, carpopedal spasm, laryngospasm, or definite convulsive seizures.

Mental development may appear to be retarded as a result of severe rickets. However, the retardation is not actual, but its simulation is dependent on delay in motor performance as compared with expected behavior for the age. Muscular activity becomes entirely normal with recovery from rickets.

Diagnosis

The diagnosis of rickets by means of the symptoms and signs already described would appear to be easy. However, in many instances the disease is present in a mild form, and mistakes in diagnosis are made frequently when the criteria used for diagnosis are not sufficiently definite. In cases of mild rickets the diagnosis is unreliable when based wholly on physical examination, without the use of roentgenograms and chemical studies of the blood. For example, no sharp dividing line exists between normal beading of the ribs and that which is slightly abnormal. No dependence can be placed on studies of the incidence of rickets when physical examination alone has supplied the criteria for diagnosis.

In cases of marked and active rickets, roentgenograms supply evidence that is completely reliable. In very mild rickets the roentgenographic changes may be equivocal and experts may disagree as to their significance. When bone is growing rapidly, minor irregularities of calcification in the metaphysis may appear in the roentgenogram. These irregularities accompanying rapid growth occur when the retention of calcium and phosphorus is maximum and are wholly normal, yet they are quite similar in appearance to the earliest roentgenographic changes that take place in rickets. In a considerable proportion of the published surveys of groups of babies to determine the incidence of rickets these minor roentgenographic changes have been interpreted as rachitic, whereas in many instances they definitely are not. Several years ago one author reported

rickets in more than 60 per cent of a group of babies under excellent supervision and receiving vitamin D in amounts that should be considered ample. At approximately the same time another author, also expert in the diagnosis of rickets, reported an incidence of 15 per cent in a group of babies selected at random at a low economic level in our largest urban center. Thus it becomes necessary to distinguish between "rickets" diagnosed radiographically by means of these unimportant bone changes and rickets that is clinically significant. Clinically significant active rickets has associated chemical changes in the blood that are in themselves diagnostic; "rickets" resulting from rapid growth is associated only with normal calcium, phosphorus and phosphatase values in the blood. Large scale radiographic surveys are much more easily made than are surveys using blood values as a criterion. It is for this reason that confusion in diagnosis has arisen. A combination of radiographic and blood studies is preferable.

When babies who have physical changes of rickets come under observation, even if the diagnosis is unmistakable, it becomes desirable to determine by radiographic and blood studies whether the rickets is still active or whether the deformities found are only the residue of rickets already healed. The subsequent management of these two states differs markedly.

Prevention and Treatment

Infantile rickets is both preventable and curable. When babies are fed appropriate amounts of either human milk or cow's milk, the intake of both calcium and phosphorus is adequate. The only other material necessary for the prevention of rickets is vitamin D. The amount of vitamin D required for the prevention of rickets and for good retention of calcium and phosphorus is discussed in Chapter VII. Rickets is prevented in most instances by 15 units of vitamin D daily for each kilogram of body weight.

This amount is supplied by one-third teaspoonful of cod-liver oil of moderate potency. Better growth and better retention of calcium and phosphorus are obtained with 50 units daily for each kilogram, an amount supplied by one teaspoonful of cod-liver oil of moderate potency or by fortified milk containing 400 units to the quart. An amount of cod-liver oil that supplies approximately 350 units of vitamin D daily is adequate throughout the period of infancy. The various cod-liver oils on the market range in vitamin D potency from 85 to 260 units to the gram, or 310 to 900 units to the (4 ml.) teaspoonful. Concentrates of vitamin D in oil are less efficiently utilized and approximately 50 per cent greater unitage is necessary than is stated for cod-liver oil. Viosterol contains 10,000 units to the gram, or approximately 200 units to the drop. Thus three to four drops of viosterol daily is a fully adequate preventive amount. Doses of ten to fifteen drops daily, commonly recommended, are detrimental (see Chapter VII). Concentrates of vitamin D are available in media that permit easy dispersion of the vitamin in milk. When such preparations are added to the formula the dosage needed is no greater than that described for cod-liver oil.

Rickets may be prevented also by exposure of the skin to sunshine or to artificial sources of ultraviolet rays. Although this is an effective means of prevention, it is not as practical in many respects as the inclusion of vitamin D in the diet. In many parts of the country the usefulness of sunshine is seasonal and at any season the amount of ultraviolet rays obtained is variable and uncertain unless the program of exposure is definitely planned. The use of artificial sources of ultraviolet rays requires not only planning, but close supervision as well. The use of ultraviolet rays offers no known advantage over the use of ingested vitamin D.

Prematurely born babies present a special problem in rickets prevention as discussed in Chapter XXVII. No

satisfactory evidence exists that the baby born prematurely requires more vitamin D than does the baby born at term, though a greater requirement has been postulated and is commonly accepted. Because of the present state of opinion and knowledge, it is advisable to administer to the prematurely born baby approximately twice as much vitamin D as has been discussed for the baby born at term. Ultimately a stage of growth is reached when the requirement is the same as for the baby born at term.

Any amount of vitamin D that will prevent rickets will also bring about its cure. However, the cure in these circumstances is slower than usually is desired. Consequently the administration of larger amounts of vitamin D is advisable until recovery from rickets occurs. The larger the amount of vitamin D given, the more quickly does the healing process begin. With the curative doses commonly employed, changes in the calcium and phosphorus content of the blood may be observed in about ten days, and radiographic evidence of beginning bone healing in about twenty days. With very large doses, blood changes may occur within three or four days and bone changes within ten days.

If cod-liver oil is the chosen source of vitamin D for therapy, three teaspoonfuls daily of a high-potency oil produce fairly rapid healing. The same result may be expected from twenty drops of viosterol daily (4000 units of vitamin D). The high-potency fish-liver oils, such as percomorph-liver oil or the cod-liver oil concentrates, also are satisfactory in a dosage similar to that of viosterol. Larger doses of these materials may be employed, if desired, but the use of such amounts should be temporary and only until healing is well under way.

The use of a single dose of vitamin D administered intramuscularly has been advocated to cover the need for an entire winter season. The amount usually employed for

this purpose is 600,000 units. Another procedure that has had some usage is oral administration of 100,000 units once each month. Even though no ill effects have been reported from either of these procedures, neither is to be recommended in our present state of knowledge. The type of observation necessary to determine ill effect has not yet been made. One might expect the same type of toxic effect as is discussed in Chapter VII for daily doses of proportionate size.

No good reason exists for the administration of salts of either calcium or phosphorus. The normal diet of the full-term infant contains an abundance of both these materials. Prematurely born infants need more calcium, phosphorus, and protein than can be supplied by human milk. The human milk feeding can be fortified with dried skimmed milk, which provides all three of these substances. Some years ago it was customary with some physicians to add small amounts of elementary phosphorus to the cod-liver oil used in the treatment of rickets. By this means a wide line of provisional calcification was produced at the point of growth of bone. This change was interpreted as beneficial. It is now known, however, that the effect produced by phosphorus is the result of toxicity and the phosphorus line produced is comparable to the bismuth line frequently observed during treatment of congenital syphilis.

Any anemia that may be associated with rickets may be expected to respond promptly to iron or iron and copper therapy. In cases of severe anemia transfusions are indicated to initiate recovery.

Many of the more moderate deformities of rickets tend to become corrected spontaneously after the rickets has healed. For the correction of some of them a year or two may be required. Some of the more severe deformities require surgical correction; this is true particularly for deformities of the legs. The surgical procedure most fre-

quently used is osteotomy, followed by placing the leg more nearly in a straight line in a plaster cast until healed. It is to be remembered that immobilization of any part of the body is accompanied by considerable decalcification of the immobilized portion of the skeleton. The skeleton should be well calcified before such therapy is undertaken. Certain deformities are not easily amenable to treatment, as for example, deformities of the ribs. However, many of these decrease considerably with the lapse of time.

CHAPTER XXX

TETANY

(SPASMOPHILIA)

Tetany is a condition characterized by neuromuscular hyperexcitability brought about by a disturbance in the metabolism of calcium and phosphorus. The nervous excitability is dependent directly on a lowered calcium content of the blood, or on a decreased ionization (or availability) of the calcium that is present, though the amount may be normal. The condition may be manifest by generalized convulsions, by characteristic spasm of the hands and feet, by spasm of the larynx, or by spasm of all three of these types. The disorder may exist in all degrees of severity, depending largely on the level of blood calcium or on its ionization.

Etiology and Pathogenesis

Any condition that produces lowering of the blood calcium or decreased availability of the calcium present in the blood may be a cause of tetany. The most common and the usual cause of tetany in infancy is the disturbance in calcium metabolism associated with rickets. The discussion of rachitic tetany is the chief purpose of the chapter, but the other causes of tetany will be mentioned.

The parathyroid glands exert a strong controlling influence over the calcium metabolism of the body. Increased activity of the parathyroid glands raises the calcium content of the blood; decreased activity lowers the calcium level. Consequently, hypoparathyroidism produces tetany. Hypoparathyroidism is almost unknown in infancy except for a transitory functional disturbance that occurs occasionally soon after birth. Uncommonly, tetany may be observed in the neonatal period. The most reasonable ex-

planation for this event is that the activity of the parathyroid glands of the mother had been increased during the latter part of pregnancy, resulting in partial suppression of the activity of the parathyroid glands of the infant. In such case, sometimes several weeks are required after birth before the parathyroid glands of the infant become fully active. The symptoms of tetany in the infant usually require active measures for control. The measures that are discussed subsequently for the control of rachitic tetany are equally effective for the control of tetany of the newborn.

Any condition that produces alkalosis may be a cause of tetany. In the presence of alkalosis the calcium of the blood is less available. The effect of decreased availability is the same as that of decreased blood content. Alkalosis may be produced by administration of excessive amounts of alkali (bicarbonate or citrate). A more common cause of alkalosis in infancy is the vomiting associated with pyloric stenosis (see p. 341). Pulmonary hyperventilation brings about increased removal of carbonic acid from the blood with a shift of the reaction toward the alkaline side. Hyperventilation sufficient to produce significant alkalosis is rarely encountered in infancy.

The availability of calcium in the blood is decreased when the phosphate content of the blood is increased. The common cause of increased phosphate in the blood is renal failure with abnormal retention of phosphate. Renal failure in infancy is most likely to be caused by congenital anomalies, such as hydronephrosis produced by partial blocking of the ureters. Such infants may have mild tetany even in the presence of moderate acidosis. The serum calcium may be normal, but the inorganic phosphorus equals or exceeds the calcium in amount and the nonprotein nitrogen of the blood is high. This condition is designated as renal hyperparathyroidism.

Some tendency exists to approach the discussion of the cause of rachitic tetany with some degree of mystery

because the mechanism is not completely clear in all instances. When babies have rickets their blood characteristically has a calcium level in the lower part of the normal range and a much decreased level of inorganic phosphorus. The first effect of administering a small amount of vitamin D is to raise the phosphate level of the blood. At the same time some of the calcium disappears, producing a lower blood level. Presumably the calcium has been deposited in the bones. If the dosage of vitamin D is adequate and its administration is continuous, its effect in lowering blood calcium is very transitory and unimportant. It is in those instances in which only a single or brief impetus has been given to the healing process that the blood calcium tends to decrease and to remain at a low level. In the natural course of events such an inadequate impetus to the healing of rickets is likely when the rachitic baby is taken out of doors for an airing on the first element day in the spring and perhaps subsequently is kept housed because of a return of unsuitable weather. The difficulty in applying this explanation of the occurrence of tetany in all instances of rachitic tetany is that sometimes the clinical evidences of rickets are meager. Despite the mildness of the rickets in a few instances, the explanation that has been given seems the most reasonable.

Many of the etiological factors pertaining to rickets pertain also to tetany. Tetany has its highest incidence late in the spring and is more common in artificially fed than in breast-fed babies. It has the same age incidence as rickets. It is more frequent in the dark-skinned than in the light-skinned races. Tetany does not occur when vitamin D has been given prophylactically in the customary manner and amount.

Infantile tetany occurs with much less frequency than formerly, just as in the case of rickets and for the same reasons. At one time tetany stood first among the causes

of convulsions in infancy. At the present time it is relatively rare, except perhaps in some of our large urban centers.

Symptoms

Tetany may be either latent or manifest. Latent rachitic tetany is that state which exists when the blood calcium has decreased below normal but not to a sufficient degree to produce manifest symptoms of the disease unless some other type of disturbance intervenes. With a blood calcium value of 7 or 8 mg. to each 100 ml. of serum, usually no symptoms of tetany are to be observed, yet on testing, increased neuromuscular irritability is found. Electrical stimuli applied over nerve trunks produce responses in the muscles supplied by the nerves with a smaller amount of electrical energy than would be required normally. An acute febrile illness occurring in the course of latent tetany is much more likely to cause a convulsion than if the blood calcium level were normal. Latent tetany responds to treatment in the same manner as does manifest tetany.

When the symptoms of rachitic tetany become manifest spontaneously, the blood calcium level is likely to be 6 mg. or less to the 100 ml. of serum. At the same time the blood phosphorus is increased to approximately the same level and blood phosphatase also is increased.

Of the three major symptoms of tetany from any cause only one, spasm of the muscles of the hands and feet, is pathognomonic of this disease. When carpopedal spasm is present the position of the hands is characteristic. The hands are flexed at the wrists and the thumbs are adducted toward the palms of the hands; the fingers are flexed at the metacarpophalangeal joints, the other joints being extended (Fig. 22). The feet are held in a position somewhat similar to that of the hands; they are flexed plantarly at the ankle, the dorsum of the foot is arched and the toes are flexed. In addition an equinovarus position may be assumed. The tonic contracture of the hands and feet may be transitory or it may continue for days at a time. In

those instances in which carpopedal spasm has not occurred spontaneously, the spasm often may be brought about by compression about the extremities with an elastic band.



Fig. 22.—Tetany. Carpopedal spasm.

This sign is elicited best in the upper extremities (Trousseau's sign). A flat rubber bandage or a blood pressure cuff is applied to the upper arm with sufficient pressure to obliterate the radial pulse for a few minutes. The hand gradually assumes the characteristic position.

Despite the carpopedal spasm often seen as a result of tetany, no general muscular hypertonicity is present. The arms and legs are freely movable, the knee jerks are not exaggerated and the Kernig sign is not present. No appearance of illness is associated with the condition. Often these children will sit quietly in bed and play with toys by the hour as best they can with stiffened hands.

Laryngospasm (*laryngismus stridulus*) is another of the major symptoms of tetany as it occurs in infancy. The adductor muscles of the larynx undergo spasmodic contraction, with the production of an inspiratory stridor. The



Fig. 23.—Tetany. Appearance during a convulsive seizure.

spasm is not sufficient to cause obstruction to expiration and no expiratory stridor is heard. The effects of the spasm become more marked when the infant cries. Laryngospasm tends to be transitory, reappearing at irregular intervals. The spasm may be sufficient to interfere with inspiration to such an extent that deep cyanosis results. Only rarely does death occur as a result of one of these attacks.

The severest manifestations of tetany are generalized convulsions. They may occur at long or short intervals. They are identical with convulsions of the cerebral type from any other cause. The infant becomes unconscious; a rigid tonic state of the body is followed rather quickly by

clonic jerkings. The hands are tightly clenched or assume the position of carpal spasm. The head is thrown back and the corners of the mouth are drawn down (Fig. 23).

The incidence of these three manifestations of tetany varies with the age of the infant. Approximately one-third of babies with tetany in the first two years of life have laryngospasm; after two years of age laryngospasm is relatively rare. The incidence of carpopedal spasm increases with the age; after two years of age approximately half of the children with tetany have spasm of the hands and feet. Convulsions occur more frequently than either of the other conditions. The incidence of convulsions is highest in the first year and gradually decreases, but it is still approximately 50 per cent in the third year. More often than not, the infant has only one of the three major symptoms. In a few instances two or even all three of the types of spasm may be exhibited by the infant; the younger the infant, the more likely are the manifestations to be multiple. After two years of age it is most unusual to find more than a single symptom.

Diagnosis

If carpopedal spasm is present, or if it can be produced by constriction about the upper arm (Trousseau's sign), the diagnosis of tetany may be made without hesitation. A finding permitting a presumptive diagnosis of tetany is a contraction of the facial muscles resulting from tapping on the face over one of the branches of the facial nerve (Chvostek's sign). This sign is much more reliable in infancy than it is a little later in childhood. In a similar manner, contraction of muscles of the leg and foot may be produced by tapping over the peroneal nerve as it rounds the fibula in the upper part of the leg.

Whenever tetany is present in either a manifest or a latent form, electrical excitability of all nerve trunks is increased (Erb's sign). In order to elicit this sign, a galvanic battery with a milliammeter is required. One

terminal is applied over the abdomen, the other over the peroneal nerve near the head of the fibula. The most characteristic reaction is obtained when the negative (cathodal) terminal is applied over the nerve, and contraction of the muscles occurs when the circuit is opened, after the passage of a current of less than 5 milliamperes. The cathodal closing and the anodal reactions are changed from the normal but are of less significance than the cathodal opening contraction. Considerable experience is required in determining the electrical reactions and in interpreting the results.

Laryngospasm with inspiratory stridor may occur as a symptom of other conditions, as for example catarrhal croup. When laryngospasm is the only symptom, tetany must be diagnosed by the various means that are discussed in this chapter.

The convulsions of tetany usually give no clue as to their cause. They do not differ from convulsions from other causes. When convulsions occur, tetany must be considered as a possible cause and tests must be made to confirm or exclude the diagnosis of tetany.

A low blood calcium value is certain evidence of tetany. Values of 6 mg. or less to 100 ml. of serum usually are associated with manifest symptoms; values slightly higher with latent tetany. In rachitic tetany the inorganic phosphorus of the blood is usually at the normal level, often a high normal, and phosphatase is increased. The blood values are the same in tetany of the newborn, except that phosphatase is not increased. In the tetany of alkalosis these various blood values are likely to be normal; the carbon-dioxide combining power is greatly increased.

In tetany of renal failure the blood calcium level may be normal or decreased. The blood inorganic phosphorus is increased to approximately the same level as that of calcium. It is the great increase in phosphorus that makes the calcium relatively unavailable. Phosphatase is not increased.

Treatment

The changes responsible for the occurrence of rachitic tetany are dependent on vitamin D deficiency. Consequently one of the early indications in the management of tetany is the administration of vitamin D. Vitamin D therapy for tetany is the same as that for rickets. The larger the dosage of vitamin D, the more quickly do the blood values return to normal. A satisfactory therapeutic dose is 2000 to 3000 units of vitamin D daily. If this dose or a larger one is used, it should be given only until normal blood values have been attained. Subsequently the amount necessary to maintain these values is small, namely, 300 to 400 units daily. After the blood values for calcium, phosphorus and phosphatase have become normal, no therapy is indicated other than the administration of the usual maintenance amount of vitamin D.

Regardless of the amount of vitamin D administered to a baby with rachitic tetany, at least several days will be required before the blood values will have reached a non-tetany level. During this period of lag the symptoms of tetany will continue unless other measures are employed to control them. Their control is important, especially in cases of convulsions or laryngospasm.

Since low blood calcium is the cause of the symptoms, the administration of calcium salts might seem to be a reasonable therapeutic measure. However, it is to be remembered that the diet already contains an abundance of calcium and more is unnecessary for adequate utilization when vitamin D is being given. The administration of soluble calcium salts by mouth causes only a slight and transitory rise in the blood level. From the standpoint of raising the blood level, the administration of calcium salts by mouth is scarcely worth while. Calcium salts administered parenterally cause a more definite increase in blood calcium. This effect also is transitory and the injections must be repeated frequently to keep tetany under control.

The inorganic calcium salts have effects in addition to those attributable to the calcium ion. These effects are discussed subsequently. Of the organic calcium salts, the lactate, acetate and gluconate are available and useful. Calcium gluconate is available in ampules in 10 per cent solution. It may be given in this strength subcutaneously, intramuscularly, or intravenously. It is relatively non-irritating, in contrast with calcium chloride, which is highly irritating to body tissues. An appropriate amount of calcium gluconate for a single injection is 10 ml. of the 10 per cent solution.

The administration of calcium chloride by mouth is highly useful in the treatment of the symptoms of tetany. The usefulness is dependent not so much on the calcium part of the salt as upon the chloride. Calcium chloride is metabolized in the body as a mineral acid. One gram (15 grains) of calcium chloride is the approximate equivalent of 75 ml. of N/10 hydrochloric acid. Previously it has been pointed out that alkalosis may cause tetany even when the blood calcium value is normal. Conversely, acidosis relieves the symptoms of tetany even when the blood calcium value is low. Presumably the shift of the acid-base balance toward the acid side leads to ionization of a larger proportion of the calcium present. The same effect as is produced by calcium chloride in the relief of the symptoms of tetany may be attained also by the administration of ammonium chloride or hydrochloric acid. If desired, hydrochloric acid may be added to the milk in the proportion of 4 ml. (1 dram) of the concentrated acid to the pint (the addition must be very slow), or the milk may be mixed with from one-fifth to one-third of its volume of tenth-normal hydrochloric acid.

Calcium chloride is administered commonly only by mouth. Solutions of this salt are highly irritating when given subcutaneously or intramuscularly. They should be given by these routes only in case of definite emergency. Calcium chloride may be given intravenously, although this

is not a common procedure. If it is administered by vein, it may be given as a 5 per cent solution in amounts up to 3 ml. for each kilogram. This amount will lower the carbon-dioxide combining power approximately 10 volumes per cent. A useful dosage of calcium chloride by mouth is 0.6 to 1.0 Gm. (10 to 15 grains) three to four times daily, depending on the age or size of the infant. The oral administration of calcium chloride is common in the treatment of infantile tetany. Its administration is begun as soon as the diagnosis of tetany is made. However, the effect in relieving the symptoms of tetany is not attained for many hours, often as much as 24 hours or more. If calcium chloride medication is the means chosen for the control of the tetany symptoms until the vitamin D has become effective, some other additional method of therapy must be employed during the first 24-hour period of management.

Magnesium has the same sedative effect on neuromuscular excitability as has calcium. Magnesium sulfate solutions have little local irritating effect and may be given intramuscularly. The effect of magnesium sulfate given intramuscularly is prompt. Within a minute or two the symptoms of tetany disappear completely. Magnesium sulfate may be injected as a 10 per cent solution of the anhydrous salt; crystalline Epsom salts contain 50 per cent water of crystallization and twice the amount must be used. The maximum dose of the 10 per cent solution should not exceed 2 ml. for each kilogram of body weight, and it is advisable not to give more than 1 ml. for each kilogram. The use of the smaller dose, with repetition if necessary, is much safer. Magnesium in excessive dose is a powerful respiratory depressant. Symptoms of respiratory failure appear almost immediately if they appear at all. If such symptoms should occur, it is most useful to remember that, while the effects of calcium and magnesium are in the same direction as regards relief of the symptoms of tetany, they are antagonistic in other respects. Parenteral injection

of any soluble calcium salt that may be at hand produces almost immediate relief from the depressant effects of magnesium.

Parathormone, the active principle of the parathyroid glands, produces a rise in blood calcium when injected parenterally. The injections must be repeated to maintain the effect. Rachitic tetany presumably is not dependent on impairment of function of the parathyroid glands. The source of the increased calcium in the blood after parathormone therapy is the body stores of calcium, the bones. Parathormone therapy is unnecessary and it would seem preferable not to withdraw calcium from the bones of a rachitic infant.

Dihydrotachysterol is used frequently to maintain the blood calcium level in cases of hypoparathyroidism. It should be useful also in increasing the calcium content of the blood in cases of rachitic tetany. However, it has not been shown that the calcemic effect of dihydrotachysterol differs qualitatively or quantitatively from the calcemic effect of calciferol or vitamin D₂. The calcemic effect of both these preparations presumably is a part of the toxic effect incident to the administration of relatively large doses. The doses required are much larger than those that have been discussed for vitamin D in this chapter. With the administration of such large doses the increased calcium of the blood is mobilized from the bones as well as from the intestinal tract. If this type of effect is desired in cases of rachitic tetany, it may be attained through the use of increased intake of vitamin D, as well as by the use of dihydrotachysterol.

From the preceding discussion it is apparent that several approaches exist to the control of the symptoms of rachitic tetany until such time as vitamin D has become effective in producing a normal blood level of calcium. Some choice of regimen must be made. While several choices exist, the following scheme is suggested. As soon as the diagnosis of tetany is made, the symptoms may be brought under

control immediately by the parenteral injection of magnesium sulfate. Calcium chloride administration by mouth then may be started. If the symptoms of tetany begin to reappear before the calcium chloride has become effective, the magnesium sulfate injection may be repeated. The effect of magnesium sulfate may be expected to last for many hours, often twenty-four hours. After the first day or at most two days, calcium chloride medication alone may be expected to keep the symptoms of tetany under control.

If a baby comes under care with convulsions and no other symptoms of tetany, a diagnosis of the cause of the convulsions must be made before specific means of therapy can be employed. The first duty to the infant is to control the convulsions. This may be accomplished by means of the various types of sedative therapy commonly employed for this purpose. With the convulsion controlled, the cause should be sought. If the cause proves to be tetany, the measures that have been discussed are applicable.

Tetany of the newborn responds to treatment identical with that described for rachitic tetany. Tetany of alkalosis ceases when the alkalosis is corrected, which may be accomplished through administration of saline solutions and if necessary the use of acidifying agents in addition, as is discussed under Pyloric Stenosis. Immediate relief from tetany can be given by an injection of magnesium sulfate.

When renal failure has attained a degree of severity great enough to produce tetany, cure of the underlying condition is no longer possible, but control of the symptoms of tetany may be attained by careful use of magnesium sulfate. Because renal failure is associated with acidosis, tetany is not common even though the levels of calcium and phosphorus are such that tetany would occur in the absence of acidosis. When acidosis of renal failure is severe, some correction of it becomes necessary, but the correction never should be complete, for tetany would then develop.

CHAPTER XXXI

SCURVY

(SCORBUTUS)

Scurvy is a nutritional disorder resulting from deficiency of vitamin C. It is characterized in part by failure of nutrition, by a tendency to hemorrhage throughout the body, especially in the bones, and by alterations in bone growth.

Etiology

Scurvy may occur at any age and in any person deprived of ascorbic acid, even though the diet and hygienic conditions may be excellent otherwise. Scurvy occurs more frequently in infants than in older persons because of the restricted character of the infant's diet. In Chapter VII some of the characteristics of vitamin C are discussed as well as the relative amounts in human and cow's milk. From the facts discussed it is apparent why scurvy is more frequent in artificially fed than in breast-fed babies. Even when scurvy was much more common than it is at present, it was rarely clinically apparent before six months of age. The reason for such a long period before the symptoms of deficiency develop is not that it takes so long to deplete the body stores and to develop abnormal changes, but rather that the food is not completely devoid of vitamin C. With complete deprivation, symptoms of scurvy appear within a few weeks. The incidence of scurvy increases after six months of age, and decreases as soon as a greater variety of foods is added to the diet. Some years ago scurvy was observed rather commonly up to the age of twelve to fifteen months. At the present time scurvy is an uncommon disease at any age. When it is observed, its occurrence usually is not to be attributed to the mother's lack of knowledge of the fact that orange juice or some

substitute should be given: often the mother concludes that orange juice has caused a digestive disturbance and she omits it from the diet without substitution of another source of vitamin C.

Pathology

The most obvious changes in scurvy are dependent on extravasation of blood through capillary walls. The hemorrhages that result are not associated with abnormalities of blood coagulation, but apparently are caused by changes in the vessel walls. These changes are not detected by the usual histological examination. They are dependent, presumably, on relative lack of cement substance between the cells of the capillary walls and perhaps on failure of growth of supporting connective tissue and deficient production of collagen. Ascorbic acid seems to be essential for growth of connective tissue—a relationship which is important also in the healing of wounds.

The most characteristic changes of scurvy are those which take place in the bones. All bones of the body are affected, but the abnormalities are most prominent and most easily detected in the long bones. In scurvy uncomplicated by rickets, provisional calcification in the metaphysis proceeds normally, but the newly calcified osteoid tissue is not converted into normal bone. In the normal growth of bone the newly calcified osteoid tissue is invaded by growing capillary tufts and becomes trabeculated by osteoclastic and osteoblastic activity. When vitamin C is deficient, the formation of new capillary tufts is retarded or stopped, and osteoblasts are greatly decreased in number and function. Thus one of the characteristics of scurvy in infancy is a broad, fairly dense line or band of provisional calcification in the metaphysis and in other areas where new bone is being formed. In the epiphysis the broadened line of calcification appears as a ring about the bone. In association with this band of increased calcification in the metaphysis and immediately adjacent to it,



Fig. 24.—Scurvy. Early radiological signs. Note ground-glass appearance of shafts, narrow dense ring round epiphyseal centers, broadened zone of provisional calcification in the metaphyses, with areas of decreased density immediately beyond, and spurs at outer edges of the metaphyses.

usually, is a narrow band of rarefaction of the bone of the shaft. The band of rarefaction is often called the scurvy line. The bone of the entire shaft is affected, but to a much less conspicuous degree than that of the metaphysis. In normal bone osteoclastic and osteoblastic activity is continuous, with the maintenance of good trabeculation. In scurvy the formation of trabeculae is impaired, chiefly because of decreased osteoblastic activity. The resulting poor trabeculation causes a homogeneous ground-glass appearance of the bone in a radiograph. For these same reasons the cortex of the bones becomes thin and the entire bone is softer than normal (Fig. 24).



Fig. 25.—Scurvy. Subperiosteal hemorrhage.

The metaphyseal ends of the bones become increasingly fragile and in advanced scurvy fracture with epiphyseal separation is common. The fractures occur with trauma that ordinarily would be negligible. They occur through the rarefied band of bone next to the metaphysis more often than through the band of increased provisional ossification.

The bone marrow also is affected in scurvy. On histological examination it may have lost much of its resem-

blance to normal marrow and may have taken on the appearance of loose connective tissue.

In advanced scurvy hemorrhages almost always are found in relation to the bones. They are found more frequently in the bones of the thighs, legs, and upper arms than in other bones of the body. The hemorrhage characteristically is subperiosteal, with its point of origin at the metaphysis. As the hemorrhage increases in size, the periosteum is stripped from the bone. These hemorrhages develop as tumor masses, with the greater part of the swelling near the joint (Fig. 25). When the subperiosteal hemorrhage first occurs, it is more difficult to detect radiographically than later, when the periosteum has formed a small amount of bone in its new location.

Symptoms

The symptoms of scurvy develop gradually and insidiously. An infant who previously may have been thriving begins to take feedings poorly. Gain in weight ceases and actual loss may occur. The infant loses his color, becomes progressively paler, is fretful, peevish and irritable, and resents handling. Little or no fever is present. One of the early signs of scurvy is an increase in the pulse rate to 150 or more and a coincident increase in the respiratory rate, but not to a proportionate degree.

The first symptom that usually arouses the definite suspicion of scurvy is tenderness of the extremities, especially of the lower extremities. The child cries out with pain when the diaper is changed or when the legs are moved. A slight degree of swelling over the tender extremities may be present. Coincident with the appearance of tenderness, a tendency to hemorrhages is noted. The skin bruises easily, even slight trauma being followed by more or less extensive ecchymosis. The gums are congested, especially along the edges adjacent to erupted teeth. If the teeth have erupted, or if they are just under the surface of the gums, hemorrhage may occur into the tissues

of the gums about the teeth, making the gums swollen and purple. A frequent location of hemorrhages is under the mucous membrane covering the hard palate.

Even in the early stages of scurvy, red blood cells may be found in the urine by microscopic examination. In those instances in which the symptoms are recognized as being indicative of scurvy and in which suitable antiscorbutic treatment is instituted, all the symptoms rapidly disappear; but if the condition is untreated, it progresses.

In a case of fully developed scurvy the symptoms are unmistakable: the affected extremities are swollen over the areas of subperiosteal hemorrhages; voluntary motion is absent and extreme pain is produced by passive movements. Hemorrhages occur, not only under the skin, but often from the mucous membranes, the nose, and the intestinal tract. The mouth may be the seat of ulcerative stomatitis with the production of a foul breath; the teeth may become loosened and may be lost. Hemorrhage from the kidneys may be sufficiently great to lead to macroscopic hematuria. When hematuria is gross, albumin, casts, and white blood cells are generally present in the urine. Some fever almost invariably is present in cases of severe scurvy; the temperature may be as high as 38.5 or 39° C. (101 or 102° F). A severe degree of anemia occurs. We have observed a red blood cell count as low as 900,000, with a hemoglobin value of 1.5 grams. Emaciation is characteristic for babies with scurvy of long duration. Sometimes the heart is found enlarged as a result of scurvy, the enlargement being dependent on myocardial weakness.

Diagnosis

It is rather seldom that other conditions must be considered in differential diagnosis in cases of scurvy. The history of deprivation of vitamin C is of value when it is definite. The statement that the baby has been receiving vitamin C is not always trustworthy. Determination of the amount of ascorbic acid in the blood is helpful, but not

necessary for diagnosis. Little or no ascorbic acid is to be found. Scurvy does not affect the calcium and phosphorus levels in the blood, but phosphatase is greatly reduced.

The hemorrhages of purpura do not have the same distribution as those of scurvy. Rarely are purpuric hemorrhages found subperiosteally in the bones, though at times they may occur in the joints. In nearly all instances of bleeding from hemorrhagic diseases other than scurvy, some defect is to be found in the blood-clotting mechanism. Henoch's abdominal purpura and Schoenlein's rheumatic purpura are exceptions, but these conditions seem not to occur in infancy.

The failure to move the extremities because of pain is spoken of as pseudoparalysis. The distinction between pseudo- and real paralysis usually is not difficult. Except for a period of hyperesthesia and muscle spasm in poliomyelitis, pain is not to be expected on passive motion in instances of actual paralysis. Pseudoparalysis is caused also by the osteochondritis of infantile syphilis. Furthermore the radiographic changes of syphilitic osteochondritis and of scurvy have features in common. It is of some assistance to know that osteochondritis of congenital syphilis always has its onset before six months of age, whereas scurvy rarely becomes manifest before the age of six months. Congenital syphilis is not associated with hemorrhages after the neonatal period. Finally serologic and therapeutic tests will remove all doubt as to which disease is present.

A disease entity that may be confused with scurvy has been described, but it has not yet received a generally accepted name. Caffey has described the disease under the name Infantile Cortical Hyperostoses. The disease is characterized by irritability, fever, anemia, leukocytosis, and changes in periosteum. The disease has been observed in a few children between two and three years of age, but chiefly it is a disease of early infancy, having its onset in

most instances under six months of age. The cause is unknown. No pathogenic organism has been found in association with the disease. Viruses have not been investigated. Because of the widespread bone involvement, the disease has been considered as possibly of metabolic origin. Fever is moderate and intermittent and the white blood cell count is variable. The baby is brought for care usually because of irritability and pain on handling. Roentgenograms of the affected bones show chiefly elevation of the periosteum. In the long bones the periosteal reaction is greater along the shafts than in the metaphyseal regions. The thickened periosteum has a laminated appearance. No scurvy line is present. Trabeculation is good and the bones do not have a ground-glass appearance. The epiphyses do not have a circumscribed line of increased density. No hemorrhages are present. Any bones may be affected, including the bones of the face. Phosphatase is either increased or normal. The disease does not respond to vitamin C, sulfonamide, or penicillin therapy. In fact, relapse may occur during intensive ascorbic acid therapy. The disease often is chronic, lasting several months, but the prognosis is good.

If scurvy and rickets should be present simultaneously, the radiographic diagnosis of scurvy may be somewhat difficult. Rickets prevents the formation of the line of provisional ossification in the metaphysis, and this line is important in the radiographic diagnosis of scurvy. In the presence of rickets it may be impossible at times to make a diagnosis of scurvy on the basis of roentgenograms alone.

Treatment

The treatment of scurvy consists in the administration of ascorbic acid or of foods containing it. Fruit juices and tomato juice are customary sources of this material. Orange juice is the most commonly used source for infants. Although 30 ml. (one ounce) of orange juice daily will cure infantile scurvy, the response is slower than if larger

amounts are given. One hundred twenty milliliters (four ounces) daily will bring about rapid relief. Synthetic or natural ascorbic acid will produce relief similarly when administered in amounts comparable to those contained in the proposed amounts of orange juice. Orange juice has an average ascorbic acid content of approximately 50 mg. to 100 ml. (15 mg. to the ounce). It is seldom that parenteral administration of ascorbic acid would be indicated; however, it is promptly effective when given intravenously as the sodium salt.

Within twenty-four hours after the beginning of ascorbic acid therapy marked decrease in pain is to be noted when the extremities are moved. In the milder forms of the disease all pain may have disappeared in this period of time. The time required for complete recovery depends on the amount of damage present. Reparative processes begin immediately, but the bones may not resume their normal appearance and structure for several weeks or even longer.

Other than the administration of vitamin C the treatment of scurvy is symptomatic. Iron medication is helpful in combating the anemia. If the anemia is severe, blood transfusion is advisable. Only rarely and in instances of most severe scurvy will orthopedic measures become necessary for the correction of bone deformities. In cases of epiphyseal separation no special treatment is indicated even though displacement has occurred. Correction occurs spontaneously during the period of recovery from scurvy.

CHAPTER XXXII

MISCELLANEOUS TECHNIQUE

Collection of Urine

The method for collection of urine in infants depends on what is desired in the way of study. For the usual routine examination of approximate acidity, albumin and sugar content, sediment, etc., special precautions in collection need not be observed. All that is desired is the collection of a clean sample. In male infants this is done by strapping the proper sized test tube in place with a cloth T-binder. Adhesive tape may be used instead of the cloth, but if several specimens are required the frequent removal of the adhesive tape will cause excoriation of the skin. The cloth binder is provided with a hole, through which the test tube is placed, and if the test tube has a sufficiently large lip it will not slip through the hole after the binder is strapped (Fig. 26).

In the case of the female infant a similar test tube may be used, fastened accurately in place with a square piece of adhesive plaster. It is better, however, to use an ordinary canary bird feeding trough. The edges of the hole in the trough are covered with adhesive tape to prevent excoriation. The trough is placed in the proper position so that when the infant is lying on her back the hole will be uppermost (Fig. 27). The trough is anchored in place with a T-binder, two arms of which are placed around the abdomen, the third passing between the buttocks, and being pinned in back to the other two. In either case, after the test tube or trough has been placed, it is necessary to prevent the infant from turning over and spilling the collected urine. For this purpose restraints are used by means of which the arms and legs are anchored firmly to the mattress.

For twenty-four-hour collection of urine from male infants a rubber glove finger and adhesive tape are substituted for the test tube and binder. The glove finger is placed over the penis and is attached to the pubes and upper portion of the scrotum by means of adhesive. The

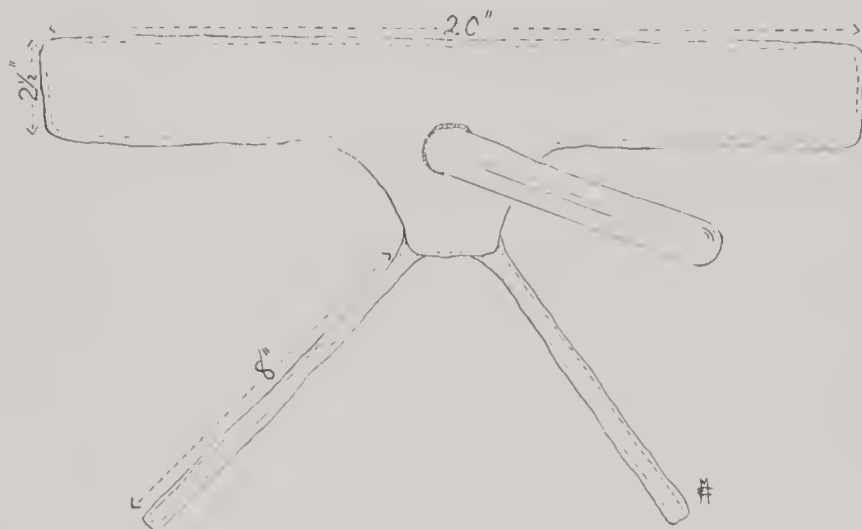


Fig. 26.—“T” binder with test tube attached for collection of urine from male infant.

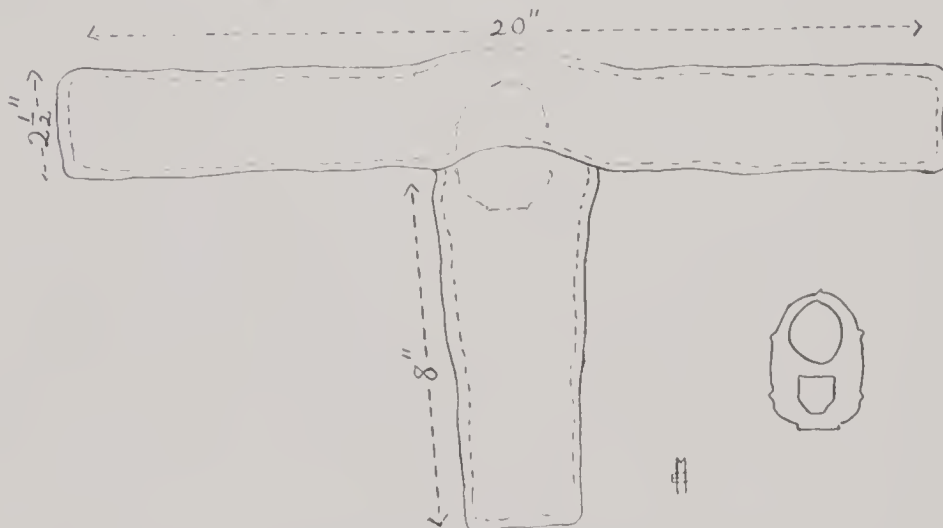


Fig. 27.—“T” binder with bird cup, arranged for collection of urine from female infant.

junction between the rubber and adhesive is reinforced outside and inside by small diamond-shaped pieces of adhesive. The tip of the glove finger is cut off and the rubber finger is joined with adhesive tape to a glass tube. The glass tube is connected with sufficient rubber tubing

to reach nearly to the bottom of the collecting bottle, which is taped to the end of the crib. The head of the crib should be raised a few inches to permit drainage. The baby's feet should be restrained with stockinet tapes, so that they do not get tangled with the collecting tubes. A few milliliters of toluol in the collecting bottle acts as a preservative.

If urine is to be collected for bacteriologic study, it is best in the case of both male and female infants to obtain specimens by catheterization. If urine is to be collected for a study that includes determination of pH, it is necessary to prevent loss of carbon dioxide from exposure to air and to prevent bacterial growth after collection. In such instances the collecting tube extends to the bottom of the bottle which contains a layer of mineral oil and a few milliliters of toluol.

Urine Cultures

A satisfactory method for determining the presence of such bacteria as colon or typhoid bacilli or staphylococci in the urine is to deposit a drop of the centrifuged sediment of a catheterized specimen of urine on a poured Petri plate of bromcresol medium, using a platinum loop, and on a second plate five to ten drops. The surface of each is smeared over with a sterile platinum wire, first spreading the single drop over its plate and then the second plate containing a larger amount of the urine. After twenty-four hours' incubation, isolated colonies when present can be picked, smeared for microscopic examination, and subcultured for identification. For such organisms as pneumococci and streptococci, plates of blood agar are used.

Stool Cultures

In general, cultural examination of the stool is limited to a search for typhoid and dysentery bacilli. To isolate these from the colon bacilli normally present in the stool, special solid culture medium containing 1 per cent of lactose and an indicator is used. Lactose-litmus-agar or lactose-

bromocresol purple-agar plates are quite satisfactory. It is essential that the stool specimen should be *fresh*. It may be obtained by inserting a sterile catheter into the rectum and collecting the material caught in the "eye" of the catheter; a sterile cotton-tipped applicator may be used instead of the catheter; or a fleck of bloody mucus from a freshly passed stool may be used. The material is emulsified in a small amount of sterile salt solution or broth, and a drop of this emulsion is placed on the dry surface of such a plate. This material is smeared over the surface of the medium with a sterile bent glass rod or wire. After incubation for twenty-four hours, the isolated colonies of bacteria which develop, if colon bacilli, produce acid from fermentation of lactose and change the color of the medium. If litmus is used, the acid changes the color to red, and if bromocresol purple is used, to yellow. Typhoid, paratyphoid, and dysentery bacilli do not ferment lactose and, therefore, do not alter the color of the indicator. Such colonies may be identified by the usual bacteriological methods.

Pinworm Ova

A simple and expeditious method of search for pinworm ova is based on the use of transparent, sticky tape. Such a tape is fastened over the end of a tongue depressor, sticky side out, and rubbed over the skin around the anal orifice. The tape is then removed, the sticky side attached to a glass slide, and examined under a microscope. The best time to examine for such ova is in the morning before the night clothing has been removed or the child has passed a stool.

Collection of Blood

The technique of collection of blood depends on what is to be studied. If only a few drops of blood are needed, as for determination of clotting time, hemoglobin, blood type or cell counts, sufficient blood can be withdrawn from the heel of infants or from the finger of children. The skin is

cleansed with alcohol or acetone and punctured to a depth of 1.5 to 2 mm. with a sterile Bard-Parker bayonet-type scalpel blade. The finger or heel is not squeezed as dilution with tissue fluid is not desirable. Excessive bleeding



Fig. 28.—Withdrawal of blood from frontanel.

can be stopped by pressure with an alcohol sponge or dry gauze. The finger should be pricked at the side of and parallel to the bone. Small children usually are less frightened by procedures that they can see than by those beyond

their line of vision; hence finger pricking is a less distressing procedure than pricking the lobe of the ear. Further cooperation often is obtained by allowing the child to choose which finger shall be pricked.

For many bacteriologic, serologic, and chemical studies, the amount of blood necessary is such as to require venipuncture. Blood may be drawn from any of the following veins, which are listed in the order of their accessibility in the infant; superior longitudinal sinus, external jugular, internal jugular, femoral, saphenous, and antecubital veins. Each source requires a special technique.



Fig. 29.—Withdrawal of blood from external jugular vein.

Blood can be withdrawn from the superior longitudinal sinus so long as the anterior fontanel is open, but blood or fluid should be introduced by this route only in an emergency. A short-beveled 20 to 22 gauge needle about 1 to 1.3 cm. long is preferred. The infant is wrapped in a sheet with his arms at his sides (mummied), and is placed flat on the table in the supine position with the head held firmly by an assistant. The scalp over the anterior fontanel is

cleansed with iodine and alcohol. The needle is inserted at the posterior apex of the fontanel, directed posteriorly at an angle of 45 degrees or less. It is inserted to a depth of only about 1 cm. in order to avoid passing completely through the sinus. After the needle is removed, pressure is applied with an alcohol sponge to check bleeding.



Fig. 30.—Withdrawal of blood from internal jugular vein.

For use of the external jugular vein a needle of fairly large gauge (18-20 usually) is selected. A needle of fairly short bevel is desirable. The shorter bevel lessens the risk of transfixing the vein which may cause extravasation of blood around the vein. The infant is mummied and is held firmly on his back with the head lower than the shoulders, either by use of a sandbag under the shoulders or by holding the head lower than the edge of the table. The head is turned so as to bring the external jugular vein uppermost. With one hand pulling down on the uppermost shoulder and with the other hand holding the head, a single attendant is able to hold the parts firmly. After cleansing the skin, the vein is entered from above. After the needle

has entered the vein for a distance of one-quarter to one-half inch, the plunger of the syringe is withdrawn slowly and blood is aspirated into the syringe. The blood is then discharged from the syringe into a suitable container.

When blood is to be taken from the internal jugular vein, the infant is restrained in the supine position and the skin prepared with iodine and alcohol. A long, 18-20 gauge, short beveled needle is inserted through the skin at a point midway between the mastoid process and the medial end of the clavicle. The needle is so inserted that it passes posterior to and just beneath the sternocleidomastoid muscle and is directed toward the median end of the clavicle. Usually the needle must be inserted about one inch. After the needle is removed, pressure should be applied with an alcohol sponge for at least a minute to prevent hemorrhage.

For withdrawal of blood from the femoral vein the baby is restrained in the supine position, the selected thigh rotated externally, adducted, and slightly flexed. The skin is cleansed as usual. The femoral artery is located by palpation just below the inguinal ligament near its midpoint. The needle, an 18-20 gauge, long needle, is inserted just medial to the artery and approximately one inch below the inguinal ligament and directed toward the ligament at an angle of 45 degrees. After withdrawal of the needle, pressure should be applied to prevent hemorrhage.

The internal saphenous and antecubital veins of infants are entered in the same manner as with older persons. No special precautions other than immobilization are necessary.

If the blood is to be used for ordinary serologic examination, such as the Wassermann test, it is discharged into a clean test tube. No special precautions are necessary. If the blood is to be used for chemical determinations wherein whole blood or plasma is specified, the tubes should contain a small amount of dry oxalate. Oxalate sufficient to prevent clotting of 15 ml. of blood is provided when 0.1 ml.

of a 20 per cent potassium oxalate solution is dried in the tube, conveniently done by placing the rack of tubes on a hot plate at low heat. If the calcium content is to be determined, oxalate must not be added to the tubes.



Fig. 31.—Withdrawal of blood from femoral vein.

If the blood is to be examined for pH or carbon-dioxide content, its exposure to the air should be prevented. Before it clots, the blood should be discharged under the surface of mineral oil in a test tube. After the blood has been obtained in the syringe, the needle may be removed from the syringe and a long spinal needle may be attached so that the surface of the oil may be reached. When blood is to be

cultured for bacteria, strict aseptic technique must be employed, including the flaming of the mouth of the flask containing the dextrose broth into which the blood is discharged.

Grouping and Matching of Blood for Transfusion

Blood grouping or matching may be carried out with as little as one quarter of a milliliter of blood. Two drops of serum are necessary and cells may be obtained by plunging a wooden applicator into the clot and then introducing the applicator into a small test tube containing 5 ml. of normal salt solution. The remainder of the clotted blood may be preserved in a refrigerator at 3° C. for future use. Cell suspensions in salt solution do not preserve well enough to warrant saving them, and a fresh cell suspension can be made from the clotted blood that has been preserved.

The practice of vein conservation in small infants is important. In order to save all possible veins for the transfusion, a sufficient amount of capillary blood may be obtained for matching.

The following method for blood matching is recommended: Two slide preparations are made: (1) a drop of the recipient's serum mixed with a drop of the donor's cell suspension, and (2) a drop of the donor's serum mixed with a drop of the recipient's cells. The preparation should be agitated in some manner. The slides may be tilted in various directions or shaken gently. After 15 to 30 minutes, agglutination or hemolytic reaction can be detected. An alternate method is to place a drop each of the appropriate serum and cell suspension in a serologic test tube, together with one drop of isotonic saline solution, and centrifuge for one minute at 500 to 2,000 revolutions a minute.

Compatibility tests performed in the preceding manner will not detect the presence of most acquired antibodies, such as anti-Rh and anti-Hr antibodies. These combine with specific agglutinogens slowly and maximally at a tem-

perature of 37° C. and require special handling as the agglutinates are much more fragile and harder to read than those formed as a result of action of anti-A or anti-B agglutinins. In these circumstances it may be well to know that when a newborn baby with erythroblastosis fetalis requires a blood transfusion, the erythrocytes of the mother, washed free of all her plasma, will always be compatible with the baby's plasma during the first ten days of life. This type of transfusion can be used even when the Rh types are unknown.

Blood Transfusion

Choice must be made between direct transfusion and indirect citrate transfusion. Direct transfusion seems to possess few, if any, advantages over the indirect method. Many institutions have adopted a system of blood bank. In the use of such a system it is necessary to give transfusions by the indirect method, and usually it is necessary also to use preserved blood. In the operation of a blood bank, blood may be drawn from relatives and friends of patients regardless of the blood type of the donor. If the blood type of the donor is not the same as that of the patient, the donor's blood is exchanged for blood of the correct type by means of the bank. Blood is drawn from the donor, typed, subjected to the Wassermann test, and preserved by refrigeration. Little evidence exists to suggest that fresh blood has advantages over preserved blood.

The indirect citrate method of transfusion lends itself to use in the infant better than does the direct method. Some of the advantages of the indirect method are as follows: (1) a sufficient quantity of blood for several transfusions may be obtained from the donor; (2) the blood may be administered to the patient without the necessity of cutting down on the vein; (3) the superior longitudinal sinus can be used if necessary; (4) the blood may be given more slowly than with the direct method, and in the absence of the donor; (5) the apparatus is simple. In general, the

indirect is preferable to the direct method. The technique for collecting blood is carried out as follows:

A simple apparatus useful for collecting blood consists of a 500 ml. flask roughly graduated (an adhesive tape marker is satisfactory) fitted with a two hole rubber stopper. Through the stopper are inserted two pieces of glass tubing, one of which extends only a little beyond the stopper, the other almost to the bottom of the flask. The longer tube is connected to the needle, the shorter to a suction apparatus. If suction is to be applied by the mouth of the operator, a trap is a necessary part of the system in order to prevent contamination of the flask with saliva. A glass tube filled with cotton makes a satisfactory trap. The tubing used for connections should be of latex or plastic tubing specifically made for transfusions. If new rubber tubing is to be used, it should be soaked in 5 per cent solution of sodium bicarbonate for 3 to 4 hours, then in 1 per cent hydrochloric acid for an equal time. It should then be thoroughly washed and allowed to stand twelve to twenty-four hours in distilled water before using. The entire apparatus is sterilized at 15 pounds (7 kg.) pressure for 30 minutes.

A sterile 3.2 per cent solution of sodium citrate is used as the anticoagulant, in a volume to correspond to 10 per cent of the amount of the blood to which it is added. The sodium citrate solution is introduced into the flask by aspirating it through the needle through which the blood must pass. For example, 50 ml. of citrate solution should be placed in the flask if 500 ml. of blood are to be drawn. The needle is placed in the donor's vein and the blood is aspirated into the flask. The flask is agitated continuously to assure a thorough mixture of the blood and the anticoagulant. When the proper amount of blood has been secured, the stopper and tubing are removed and the blood is filtered through several layers of sterile cotton gauze or a plastic or stainless steel mesh filter into another sterile



Fig. 32.—Blood transfusion. Apparatus for giving small transfusions using a 50 ml. syringe as reservoir for blood.

flask, which is stoppered with a sterile cotton plug. Blood so obtained may be kept in a refrigerator (3° C.) for as long as a week.

For giving small transfusions of less than 100 ml., a simple apparatus can be made from a 50 ml. syringe with the plunger removed (Fig. 32). A glass flow-indicator is inserted in the delivery tube so the rate of flow can be controlled. It is convenient to have a small glass adapter at the end of the tubing; to this is connected a short piece (about 35 cm. or 14 in.) of fine tubing about 3 mm. inside diameter. The glass needle-adapter is connected to the fine tubing. The fine tubing is more flexible and thus more easily manipulated than the heavier tubing extending from the flask. Ease of manipulation is important in placing the needle, particularly into the small veins of the scalp, hand, or foot (Fig. 33).

Sufficient physiological saline solution is poured into the syringe so that the tubing and needle are filled and about 5 to 10 ml. can be seen in the reservoir; the tube is then clamped. Ringer's or other solution containing calcium should not be used because it will cause clotting of the blood. When the vein is entered, blood will be forced back into the tubing by venous pressure, and can be seen through the glass needle-adapter. In some instances it may be desirable to use a small syringe as a handle for the needle in entering the vein; after entry the syringe is removed and the needle fitted onto the adapter of the transfusion set. After it is certain that the vein has been entered fairly, the clamp is removed from the tube and the fluid is allowed to flow into the vein. The needle is fixed in place with strips of adhesive tape. When the rate of fluid flow has been adjusted satisfactorily, the blood is poured into the syringe and allowed to flow into the vein. A second portion of saline solution is used to rinse the tubes, insuring that the baby receive all the blood. Blood for transfusion should not be warmed to body temperature prior to admin-



Fig. 33.—Blood transfusion. A closer view of the infant in Fig. 32 showing the method of inserting a length of fine tubing between the needle and the rubber tubing which is connected to the reservoir.

istration, as this procedure may cause changes in the blood to which systemic reactions are attributed.

For administering larger amounts of blood a 2-flask apparatus is useful. One variety of such apparatus is shown in Fig. 34. One flask contains blood, the other, saline solution which is used in the manner previously described for starting and completing the transfusion. Usually little hydrostatic pressure (12 to 24 in.) is necessary to cause the blood to enter the vein at a desirable rate. If the child is crying vigorously, however, the venous pressure during the prolonged expiratory phase of respiration may increase to such a point that the blood will flow in the wrong direction. In such instances, and also when a very small needle is used, it may be necessary to use several feet of hydrostatic pressure. An adjustable standard is useful for this purpose.

In general, the dosage of blood is 15 to 20 ml. for each kilogram of body weight, given at the rate of 1 to 2 ml. a minute. A convenient indicator for determining the speed of flow is a Murphy drip bulb. By means of such a device the rate of dropping of the blood may be observed. A less accurate indicator can be fashioned by placing a small amount of sterile salt solution or 70 per cent alcohol in the bottom of the glass tubing through which the air enters the flask as the blood leaves it. This solution is sucked up the tubing at the same rate that the blood is flowing out. As it is sucked up, it gradually coats the walls of the glass tubing until finally no column of fluid remains. The fluid then flows down the wall by gravity and collects again at the bottom of the tubing, whence it is sucked up as before.

When the indication for transfusion is a reduction in the blood volume due to hemorrhage (or shock), the suggestion of 15 to 20 ml. of blood for each kilogram of body weight does not apply. The amount to be given should be comparable to that which has been lost. When dealing with cardiac disease or pneumonia, the suggested amount of blood may be given, but the rate of flow should be markedly



Fig. 34.—Blood transfusion. Use of two flasks, one containing blood, the other, normal saline solution.

reduced. In a high proportion of occasions when transfusion is indicated in infancy, an indication exists also for other fluids. Thus it is frequently desirable to dilute the blood with at least an equal volume of physiologic salt solution. Ringer's solution must not be used because of the likelihood that clotting will occur as a result of the presence of calcium in the solution. When a transfusion is contemplated for a baby with marked dehydration, it should be preceded by the administration of salt solution (see p. 304).

The small veins of the scalp may be used for transfusion in infants under one year of age. The firm surface of the skull makes a good support for the needle. A hypodermic needle with a long bevel adapts itself well to this use; particularly excellent is the Bard-Parker type ($\frac{5}{8}$ inch or 1.5 cm. long and 25 gauge), since the shank is flattened. When blood has been strained carefully through a wire strainer in which are placed several thicknesses of gauze sponges, it can be expected to run through such an apparatus by gravity. The flask is elevated two to three feet above the level of the vein (Fig. 35). If desired, a special apparatus may be arranged in order that the blood may be pumped into the vein. A 20 ml. syringe is used which is equipped with a three-way stopcock. The tube from the flask of blood is attached to the side arm of the stopcock. It is best to use a short piece of rubber tubing with an adapter for the needle, as direct attachment of the needle to the syringe makes its manipulation awkward. Blood can then be drawn into the syringe from the flask and finally forced into the vein after the stopcock is turned.

The scalp is shaved over the area where a vein can be seen (usually the frontal or temporal regions) and the area is painted with iodine and alcohol. About 10 ml. of physiological salt solution are drawn into the syringe and the vein is entered. Because of the size of the vein, withdrawal of blood may not be possible, so the operator makes sure the needle is in the vein by injecting a small amount



Fig. 35.—Blood transfusion. Set-up without flask of normal saline solution.

of the salt solution. If the needle is outside the vein, a small lump will appear at the end of the needle. In this case another puncture must be made farther down. After one is sure the vein has been entered, the transfusion is carried out by one of the two procedures described above. Great care must be used in holding the needle securely in place during the manipulations.

In some instances no superficial veins large enough to permit transfusion can be seen in small athreptic infants, especially when dehydration plays a prominent rôle in the disease. In such circumstances the problem often may be solved by the use of an external jugular vein. The infant is "mummied" securely, and a sandbag is placed under the shoulders. The veins in the neck usually become distended so as to be visible when the infant is made to cry. Looseness of the soft tissues of the neck region often causes the operator to transfix the vein with the consequent production of a hematoma. If the vein has been transfixed, the needle should be withdrawn quickly at the earliest evidence of swelling, and pressure should be applied. At times, after transfixation the external jugular vein becomes prominent and can be entered at the second trial (at a lower level) with ease.

The one indication for use of a particular vein is its accessibility. Often the antecubital vein is sufficiently large and visible to be entered. At times an adequate vein may be found on the dorsum of the foot or hand, or on the mesial aspect of the ankle. When other veins are not available the femoral is sometimes used. It is often surprisingly easy to strike this vein even though it cannot be seen. In infants not more than two or three days of age it is often possible to enter the umbilical vein easily.

When no veins are easily accessible, transfusion may be given by way of the bone marrow (Fig. 36). This route carries somewhat greater risk of infection than the intravenous and strict aseptic technique must be maintained. A special 18 gauge needle with a flat bevel and a stylet

is used. Lumbar puncture needles may be substituted. The two best sites in the infant are the lower third of the femur and the upper end of the tibia. If the tibia is used, the site of injection is just below the tuberosity which lies anteriorly midway between the median and lateral condyles, on the flat medial aspect of the bone. If the femur is used, the preferred site is in the midline on the anterior

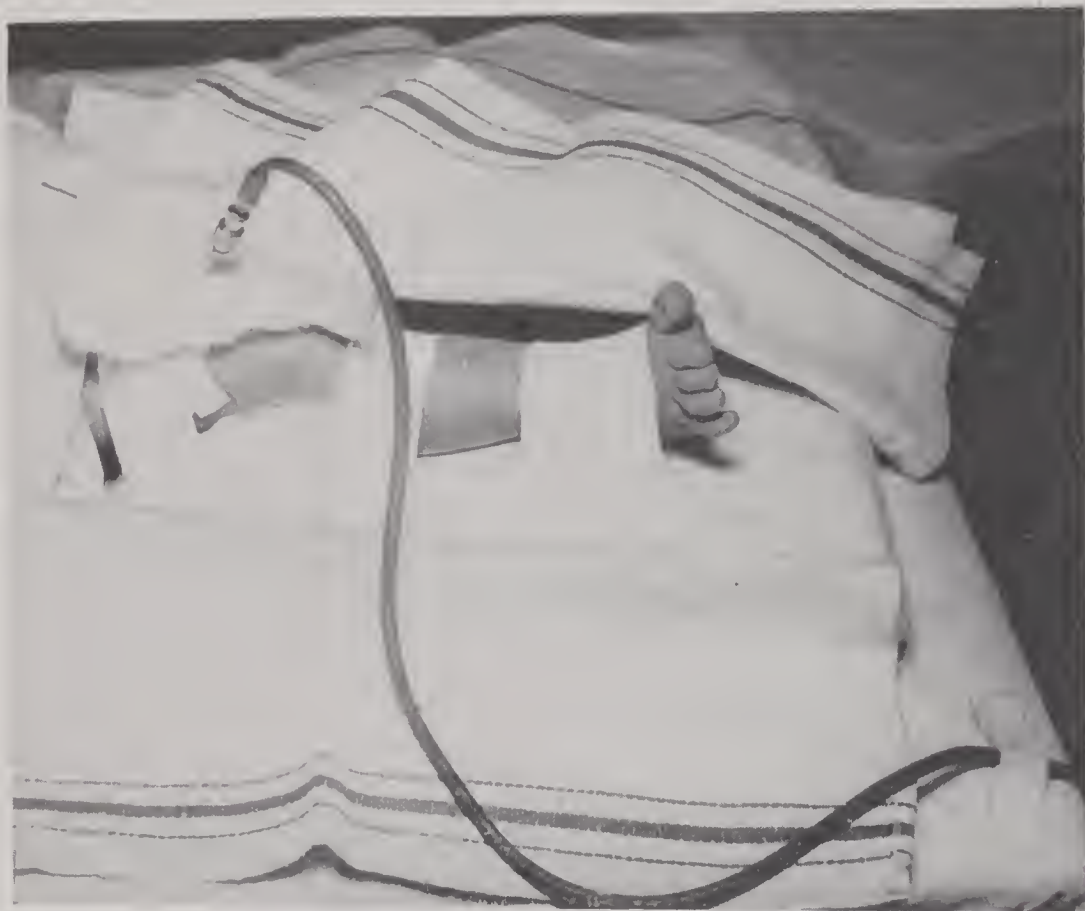


Fig. 36.—Blood transfusion into the bone marrow.

surface one inch (2.5 cm.) above the lateral condyle. The needle is always directed away from the epiphysis. The skin is prepared by cleansing with iodine and alcohol and the area is draped with sterile towels. The operator wears sterile gloves. The needle is inserted at the selected site, using firm pressure and, after the bone is reached, a rotary, not side to side, motion. When the medullary cavity is reached, the amount of resistance is sharply decreased and grating will be felt. As soon as the needle is in the

medullary cavity, the stylet is removed, but kept sterile. A syringe is attached to the needle and a small amount of marrow is removed to make certain that the needle is in the marrow cavity. With a fresh syringe saline solution in the amount of 5 to 10 ml. is injected. The fluid should enter the cavity with very little pressure. The flask containing blood is then connected to the needle by means of a glass adapter or a special curved metal tube designed for this purpose. The same precautions are used to avoid introduction of air or blood clots as for intravenous transfusion. The blood is allowed to flow into the marrow cavity by gravity or by means of a syringe, but the rate should not be more than 4 ml. a minute. After the transfusion is started, sterile sponges or towels should be placed around the needle. Immobilization of the infant should be adequate to prevent movement of the leg and displacement of the needle; blood flow into subperiosteal or tissue spaces may cause necrosis. Should swelling of tissues at the site of injection be noted, the transfusion must be discontinued. After the needle is removed, the wound is covered with a sterile dressing. The same bone should not be used within twenty-four hours after completion of an infusion. Bone marrow transfusion is contraindicated in general sepsis and in osteomyelitis. It is not advised for infants with erythroblastosis or congenital anemias.

In babies with open anterior fontanel the superior longitudinal sinus is easily entered by a needle. Relatively little danger is attached to the use of the longitudinal sinus for the purpose of withdrawing blood. Even if the sinus should be transfixated accidentally by the needle, little danger of intracranial hemorrhage exists. The danger of administering fluids by this route is definite. Even those who seemingly are expert in the procedure usually have difficulty if they persist in the practice. The injection of blood or other material over the cortex of the brain produces serious, often fatal, results. Consequently, it seems

best to recommend that fluid be given by way of the longitudinal sinus only in emergencies and in desperate circumstances.

Saline Administration

Salt solutions frequently injected are: 0.85 per cent sodium chloride; Ringer's solution; Hartmann's lactate-chloride solution; sixth molar sodium lactate solution. In the case of the first two solutions, tablets of the dry salts may be obtained from pharmaceutical firms and may be dissolved and diluted to the proper volume with distilled water. Hartmann's solution and molar lactate solution are obtainable in ampules and require only dilution with sterile water.

Saline solutions may be given intravenously into any of the veins discussed in the preceding section. Also they may be given subcutaneously or intraperitoneally. When infants are severely dehydrated or when they are *in extremis* from any cause, the circulation is greatly impaired, and as a consequence absorption from the peritoneum and subcutaneous tissues is likely to be slow and ineffective. In these circumstances intravenous administration of fluids is greatly to be preferred.

Subcutaneous Injection

Physiological saline solution, Ringer's solution, sixth molar sodium lactate, Hartmann's solution, and Darrow's solution can all be given subcutaneously. Glucose is best not given by this route as this solution may be irritating; it may be so given when necessary in mixtures of equal parts of 5 or 10 per cent glucose solution and saline or Ringer's solution. The amount of any fluid given subcutaneously at one injection should not exceed 35 ml. for each kilogram (15 ml. for each pound) of body weight. This route is not to be recommended in severe dehydration or shock as fluids in these conditions will not be drawn into the circulation satisfactorily.

In infants the two best sites for subcutaneous injections are the back and the thighs. The skin is cleansed with iodine and alcohol, a fold of the skin is picked up and a long 19 to 20 gauge needle is inserted into the subcutaneous tissue. The fluid may be introduced from a 50 ml. syringe with the needle inserted its full length. The needle is withdrawn almost to the skin and reintroduced in a different direction after 3 to 8 ml. have been injected. The skin becomes elevated, but should not become blanched. If desired, the fluid may be given by gravity as for transfusions. The needle is then held in place by a strip of adhesive tape. Two needles may be used, one on either side of the back; the needles are connected to the same flask by means of a Y tube and rubber tubing. The areas used for injection should be draped with sterile towels. The same techniques are used when fluid is given in the lateral aspect of the thighs, except that less can be given at one time by syringe into the thigh than into the back. After removal of the needle the wound may be covered with a small amount of collodion.

Intraperitoneal Injection

Before intraperitoneal injection is attempted, any abdominal distention should be relieved. The bladder should be emptied.

The technique for the intraperitoneal administration of fluids is as follows: The sterile saline solution in a flask (such as was described above for the giving of blood) is warmed to body temperature. A needle of 16-18 gauge and one and one-half inches in length, with a short bevel, is attached to the adapter at the end of the tubing. After the air is expelled from the system, the operator grasps the shank of the needle between the thumb and forefinger, at the same time clamping off the rubber tubing with the third finger and thumb. The infant is held flat on his back, the thighs immovable and extended. The skin midway between the symphysis pubis and umbilicus, previously cleansed

with iodine and alcohol, is picked up with the left hand. The needle is thrust in at an angle as acute as possible to the plane of the abdominal wall (i.e., almost parallel with the abdominal wall) and in the sagittal plane. After the needle has entered the peritoneal cavity, it is thrust up to the shank and held firmly in position with a strip of adhesive plaster. The needle should lie almost parallel with the abdominal wall. It usually then can be palpated very distinctly through the umbilicus. Saline solution is run in by gravity, until the abdomen begins to feel distended. Sometimes three to four hundred milliliters may be injected within ten to fifteen minutes into the peritoneal cavity of an infant weighing ten pounds (4.5 kg.).

Entrance may be made into the abdomen through the rectus muscle lateral to the midline. This has been recommended in the case of very young or immature infants in whom the hypogastric artery may not have been obliterated. There is perhaps less danger of puncturing the bladder in this way and in the case of very undernourished infants with thin abdominal walls, somewhat less danger of leakage after the needle is withdrawn.

The chief contraindications to intraperitoneal injection are inflammation of abdominal organs, abdominal distention, respiratory embarrassment, and contemplated intra-abdominal operations.

Dextrose (Glucose) Administration

Dextrose solutions are best given intravenously, as irritation occurs when such solutions are injected subcutaneously or intraperitoneally. The irritant effects are, however, minimized if the dextrose is combined with Ringer's solution. A mixture of equal parts of 10 per cent dextrose and Ringer's solution may be injected subcutaneously and is occasionally used when it is desired to give an extra amount of dextrose to undernourished infants when other means of administration are difficult. It is best to dispense en-

tirely with intraperitoneal injections of dextrose because of the distention which so frequently follows.

Dextrose solutions may be prepared from the concentrated solutions supplied in ampule form by pharmaceutical manufacturers. Such solutions are diluted with freshly distilled water before injection. The solutions may also be prepared from chemically pure dextrose. Weighed amounts of dextrose are sealed in glass ampules and sterilized by boiling in a water bath for 30 minutes on each of three consecutive days. Solutions are prepared immediately before use by emptying the contents of the ampule into the proper amount of freshly distilled and boiled water.

Fluids for Parenteral Use

FLUID	QUANTITY MAXIMUM	ROUTE	RATE	INDICATIONS
Saline (0.85%) or Ringer's sol.	35 ml./kg. or 15 ml./lb.	Subcutaneous Intravenous Bone marrow Continuous venoeclysis Intraperitoneal	As fast as absorbed 1 to 2 ml./ minute 4 ml./kg./ hr. Rapidly	Dehydration, acidosis, alkalosis
M/6 sodium lac- tate in water, Ringer's or sa- line or Hartmann's sol.	2 ml./kg. or 1 ml./lb. for each volume per cent rise needed	Intravenous Subcutaneous	As for saline	Acidosis, de- hydration
Sodium bicar- bonate 4%	0.5 ml./kg. for each volume per cent rise needed	Intravenous only		Severe acidosis with CO ₂ less than 20 vol- umes per cent
Darrow's so- dium-potassium- chloride-lactate- sol.	80 ml./kg. or 36 ml./lb. in 24 hours	Subcutaneous Intravenous	7 to 10 ml./kg./ hour	Severe diarrhea Renal function must be ade- quate
Dextrose 5% in water or mixed with equal parts of saline or Ringer's sol.	35 ml./kg. 15 ml./lb.	Intravenous Bone marrow Continuous venoeclysis	1 to 2 ml./ minute 1 to 2 ml./ hour	Ketosis, dehy- dration, intra- venous feed- ing

(Continued on next page)

FLUID	QUANTITY MAXIMUM	ROUTE	RATE	INDICATIONS
Blood	20 to 30 ml./kg. 10 to 13 ml./lb.	Intravenous Bone marrow	1 to 2 ml./ minute	Anemia, malnu- trition, shock
Plasma	As for blood	As for blood	As for blood	Hypoprotein- emia, shock, malnutrition
Amino acid 5% in 5% glucose	20 ml./kg. 10 ml./lb.	Intravenous Subcutaneous	1 to 2 ml./ minute	Parenteral feeding

The total amount of fluid needed by a baby in 24 hours is approximately 150 ml. for each kilogram (2.5 ounces for each pound). The basic chloride requirement for 24 hours is supplied by 100 to 150 ml. saline solution (approximately 1 Gm. of sodium chloride). More should not be given except in case of chloride deficiency, as alkalosis from vomiting or continued loss of chloride through Wangenstein drainage or diarrhea. In severe diarrhea the amount of chloride given should be $1\frac{1}{2}$ to 2 times the basic requirement. The chloride content of blood or plasma is approximately one-half that of saline solution; that of Darrow's solution 70 per cent. The remaining fluid requirement should be supplied by fluids that do not contain chloride; these can be glucose, lactate or amino acid solution as indicated, when all fluids are being given parenterally. When babies are being maintained completely by parenteral alimentation, the basic energy and protein requirement may be considered as 50 calories and 2 Gm. respectively for each kilogram each 24 hours.

When sodium bicarbonate is used, it is best to give only sufficient to increase the plasma CO_2 no higher than 40 volumes per cent. As a dehydrated baby rehydrates, base may become available from the tissues in sufficient amount to produce alkalosis if the plasma CO_2 has been restored fully to normal.

When babies are receiving partial or total parenteral therapy, a special chart for recording amounts of the various fluids is a convenience. The chart should be devised to show the total intakes of fluid, calories, protein, potassium chloride and sodium chloride. It should contain a column for each of the following: foods and fluids given orally; bicarbonate and lactate solutions; dextrose in water; dextrose in salt solution; salt solution; Darrow's solution; plasma and blood; amino acid-dextrose solution. The caloric value for each milliliter of solution is as follows: 5 per cent dextrose, 0.2; 10 per cent dextrose, 0.4; 5 per cent amino acid solution, 0.6; plasma and blood, 0.2. The protein value for each milliliter of plasma, blood and amino acid solution is 0.05 gram.

Continuous Intravenous Injection (Venoclysis)

Continuous intravenous injection of fluids is used at times in the treatment of infants suffering from severe dehydration (anhydremia) and those in a state of severe malnutrition (athrepsia). The solutions ordinarily given are 5 per cent dextrose solution, or a mixture of equal parts of 5 per cent dextrose and physiologic salt solution, or Ringer's solution. The 5 per cent dextrose solution is used

when it is desired especially to supply additional food in the case of undernourished infants or of those who for any reason are unable to take much food by mouth. It is used also to complete the fluid requirement after the basic requirement for chloride has been met with saline solutions. In some instances amino acid solutions are given for their protein value. The Ringer's-dextrose mixture is used especially to restore fluids in the presence of anhydremia. The addition of suitable amounts of the B vitamins and ascorbic acid to the fluid being administered is advantageous if insufficient of these is being taken by mouth.

The rate of injection of the 5 per cent dextrose solution ordinarily should not be greater than 4 ml. for each kilogram of body weight in a one-hour period. The dextrose or dextrose-saline mixtures may be given more rapidly during the time that dehydration is severe, the rate being decreased as the dehydration is overcome. At the beginning of the treatment the dextrose-saline solution is given at the rate of 4 to 8 ml. an hour for each kilogram of body weight; this rate is equivalent to 1 to 2 drops a minute for each kilogram. The injection of either fluid may be continuous for several days; during the period of injection the infant's entire fluid requirement and an important portion of the energy and protein requirement may be met without recourse to oral administration of food or water. It is important to bear in mind that when serum proteins are reduced, as they frequently are in prolonged illness, edema is likely to result if fluid is given too rapidly or in too large quantity or if the chloride requirement is exceeded.

The apparatus used for injection is the one described as appropriate for blood transfusion. The solution to be injected need not be warmed initially or maintained at or above room temperature. It may be expected to be at room temperature by the time it reaches the vein. No harm is caused by the slow injection of solutions at room temperature.

When superficial veins are easily accessible, or for the expert operator, the exposure of a vein by incision is unnecessary. The procedure is carried out as has been described for transfusion. In all instances the needle must be attached firmly to the skin, usually by adhesive. In those instances in which exposure of the vein becomes necessary, the following procedure is applicable.

One of the surface veins of the upper or lower extremity is cut down on and ligated on the distal side. A slit is cut in the vein and a piece of sterile ureteral catheter about three inches long is inserted and tied in. Into the catheter the needle at the end of the delivery tube is inserted. The needle should fit snugly. Before inserting the needle, the tube and needle, of course, must be filled with the injection solution. After insertion of the needle the spring clamp on the long tube is released and the rate of flow is adjusted by the screw clamp. For the first fifteen to twenty minutes, fluid may be run in about as rapidly as it will pass through the needle. The flow is then reduced to the desired rate by adjustment of the screw clamp. The rate of flow is determined by the number of drops a minute, and also by the total volume of fluid leaving the graduated cylinder each hour.

Gavage

The infant is wrapped in a sheet and held on his back. In the case of the small infant without teeth, a fairly stiff rubber catheter, attached to the barrel of a sterile glass syringe or a small funnel, is thrust into the mouth and down the esophagus. In the case of older infants, the catheter may be inserted through the nose. As the catheter enters the stomach the air bubble usually present there can be heard as it is expelled through the tube. Accidental passage of the catheter into the larynx is extremely difficult. However, if the catheter has not entered the esophagus, but has entered the trachea instead, there will be no doubt of the fact, as the infant will become suddenly very dyspneic

and cyanotic. When the air bubble in the stomach has been expelled, the glass syringe is filled with the material to be fed, and the flow is started by "milking" the catheter. The fluid to be given is run in as rapidly as possible by gravity. Before withdrawing the catheter, it is pinched tightly and is then withdrawn as quickly as possible in order to avoid reflex vomiting.

History Taking

Emphasis on the value of a good medical history seems scarcely necessary. In institutional practice a printed guide for obtaining the history serves a useful purpose, particularly for the less experienced members of the resident staff. An outline suitable as a guide for recording the medical history of infants follows.

OUTLINE FOR HISTORY TAKING

History taken by ----- from (mother, father, aunt, patient).

Chief Complaint:

Present Illness: A connected, concise, running account of the illness with date and type of onset and order of appearance and character of symptoms. Ask about those referable to respiratory and gastrointestinal tracts particularly, and skin eruptions. Any disease suggested by the information received should cause inquiry regarding all symptoms characteristic of that disease. Negative history should be noted only in relation to differential diagnosis of such suspected disease. Until one becomes expert it is well to make preliminary notes and from them condense and arrange the story of the illness on the record sheet.

Past History:

Infants (a) General: A short summary of the baby's life since birth, including such items as weight and condition at birth; presence of malformations, cyanotic attacks, convulsions or paralyses; colds or other illnesses. Immunizations. (b) Developmental: Age when sitting alone, standing, walking, talking. Dentition. (c) Feeding: Type, amount and intervals with changes and reasons for them. Any orange juice? Cod liver oil? Other foods. Medical supervision? Thrived? Character and frequency of stools.

Family History: Age and condition of health of parents and other children. Contact or exposure of patient to known or suspected tuberculosis. Has either parent had a Wassermann test ("blood test") or had any anti-syphilitic treatment ("shots for blood disease")? Miscarriages, stillbirths, or living children dead in early infancy? Allergic manifestations (asthma, urticaria, hay fever, sneezing, eczema) in maternal or paternal kin.

Interval History: If the patient has visited the Pediatric Clinic previously and returns later with a new complaint, make a short note of the health between visits before writing of the present illness.

Physical Examination:

General: Nutrition and development. Apparent mentality. Evidence of distress. Toxicity. Condition of musculature.

Skin: Eruption (character and distribution). Cyanosis, pallor, mottling, abnormal perspiration. Turgor. Scars and birthmarks or malformations. Elasticity. Palms and soles (eruption, thickening, desquamation).

Head: Scalp (nits, ringworm). Abnormal shape and size; record the circumference. Fontanel (tense, full size), craniotables. Occipital lymph nodes. Rigidity of neck.

Ears: Mastoid tenderness, aural discharge. Examination of drum and canal if indicated. Deafness.

Eyes: Blepharitis, bulbar or palpebral inflammation. Movements (ptosis, strabismus, nystagmus). Cornea.

Nose: Obstruction. Discharge (character).

Mouth and Pharynx: Teeth, number and condition (caries, hypoplasia). Gums (inflammation). Tongue, buccal mucosa (stomatitis, enanthem). Pharynx (condition of mucosa, lateral pharyngitis). Tonsils (size, character, inflammation). Congenital malformations (cleft palate). Cervical lymph nodes.

Chest: Expansion, symmetry. Rachitic deformities (rosary, flaring costal margins, Harrison's groove, other bony elevations or depressions).

Heart: Apex impulse and force, character of sounds, arrhythmia? Cardiac enlargement? Murmurs? Sounds at base.

Lungs: Type of respiration. Abnormalities (inspection, percussion, auscultation).

Abdomen: Conformation (scaphoid, distended, flat). Tenderness or rigidity. Masses or fluid. Consistency. Palpable viscera (liver, spleen, kidney). Hernias.

Bones and Joints: Posture (round shouldered, lordotic). Paralyzes, atrophies, flatfeet, spinal curvature.

Reflexes:

Genitals: Phimosis, preputial adhesions, hydrocele (cord, tunica vaginalis), inguinal hernia. Cryptorchidism. Vaginal discharge. Inguinal adenitis.

Laboratory Examinations: Urine, blood or purulent secretions are to be examined and throat cultures taken, when indicated.

Summary: Essential abnormalities found, with provisional diagnosis.

The Clinical Chart

In the medical care of infants a need exists for a graphic chart that differs in several important respects from the charts commonly used in the care of older children and adults. The nutrition of the infant is watched more closely day by day. To this end greater attention than in the case of the older child is given to the body weight, to the type of food, to the character of the stools, and to gastrointestinal symptoms. Many types of clinical charts for use with infants have been devised, but all have in common the emphasis on nutritional factors. The chart illustrated herewith exemplifies the general type of record of information considered most useful. Except under unusual circumstances, the pulse and respiration rates are not recorded in infancy. In the use of these graphic records it is customary to chart the temperature in black and the weight in red. When vomiting occurs, it is desirable to have a record of both the time and the approximate amount. In recording the bowel movements, the stools passed during the day and the night of each day are recorded separately, as this serves to give a better idea as to the progress of the infant. Either signs or abbreviations may be used to record the character of the stools. The designations used in some hospitals are as follows:

	Normal stool
/	Moderately loose stool
—	Watery diarrheal stool
^	Soft stool with mucus
^	Soft stool with mucus and pus
^ ^{bl}	Soft stool with blood (the sign may also be made in red to indicate blood)
□	Hard, constipated stool

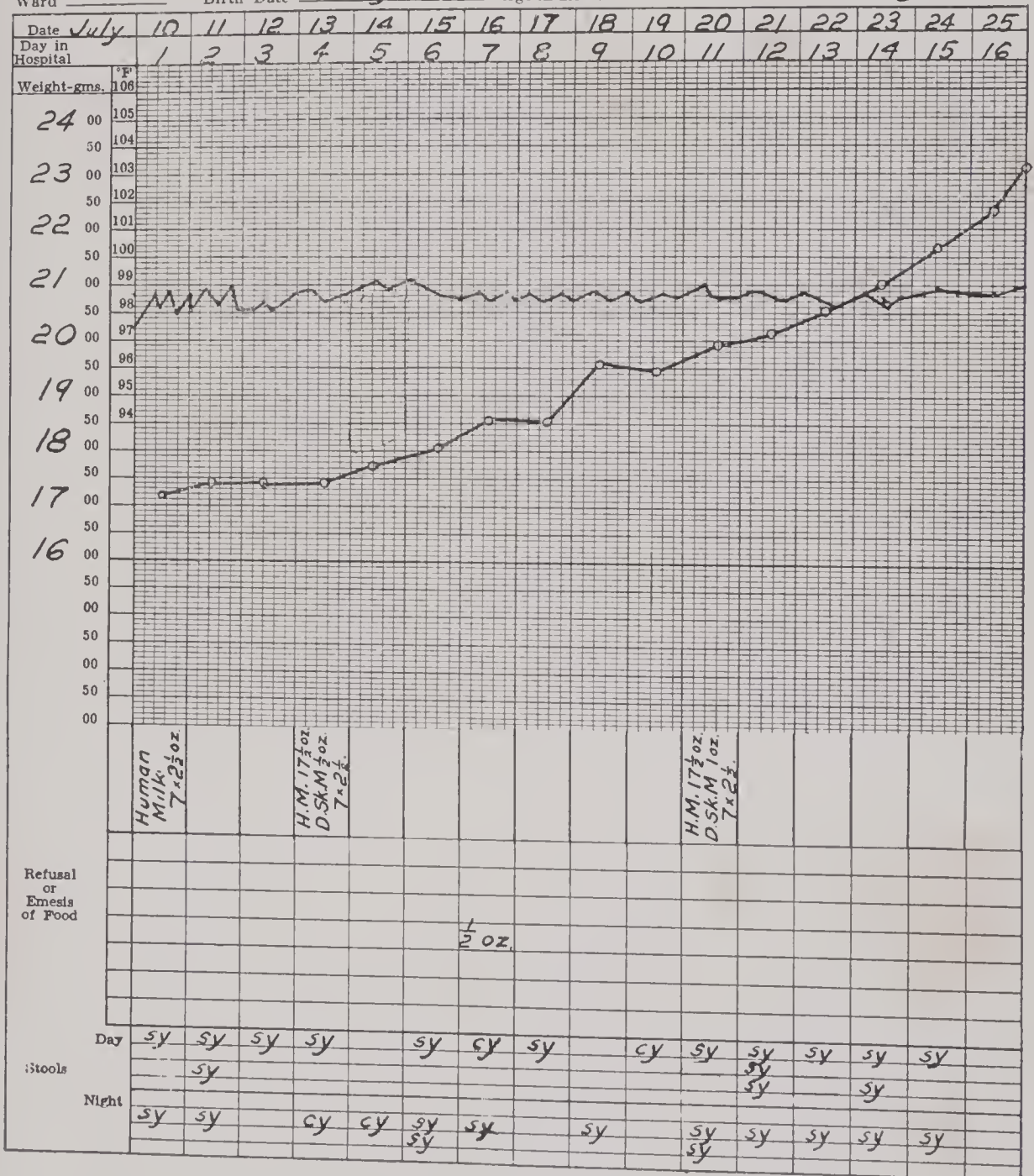
If, instead of signs, it is desired to use abbreviations, the following designations serve well:

S	Soft
F	Formed
L	Liquid
C	Constipated
Y	Yellow
B	Brown

G	Green
Bl	Blood
FS	Formed Soft
SY	Soft Yellow
LG	Liquid Green

NUTRITION - TEMPERATURE CHART
For All Children Under 2 Yrs. of Age

Name R.H. Hospital No. 41-39271 Service Pediatric.
Ward F Birth Date May 8 Age in months 2 Admission Date July 10.



[illegible]

[illegible]

OVER

BINDING LINE

Laboratory Sheet—reverse

Laboratory Sheet

A laboratory record suitable for the infant does not differ greatly from one suitable for the older person. Greater attention is given to immunization procedures and tests in the infant and child than in the adult. A record of these procedures on the laboratory sheet is helpful. A convenient form of laboratory sheet to be attached to the patient's record is shown on pp. 492 and 493.

Drugs

	DOSES		
	6 MONTHS	1 YEAR	
Acetyl salicylic acid (aspirin)	30 mg. ($\frac{1}{2}$ gr.)	60 mg. (1 gr.)	Every 4 hours
Adrenalin. See Epinephrin			
Ammonium chloride (to acidify urine)	0.3 Gm. (5 gr.)	0.3 Gm. (5 gr.)	Every 4 hours
Amytal sodium (Hypo)	30 mg. ($\frac{1}{2}$ gr.)	60 mg. (1 gr.)	Every 4 hours. More for convulsions
Antipyrin	30 mg. ($\frac{1}{2}$ gr.)	60 mg. (1 gr.)	Every 4 hours
Atropin sulfate (Mouth)	0.1 mg. (0.002 gr.)	0.15 mg. (0.003 gr.)	Every 4 hours
Belladonna, tincture	0.12 ml. (2 min.)	0.2 ml. (3 min.)	Every 4 hours
Benadryl	4.5 mg. for each kilogram (2 mg. for each pound) divided over 24-hour period		
Caffeine sodiobenzoate (Hypo)	60 mg. (1 gr.)	0.1 Gm. ($1\frac{1}{2}$ gr.)	P. r. n.*
Calcium chloride (Mouth)	0.6 Gm. (10 gr.)	0.6 Gm. (10 gr.)	3 to 4 times daily
Calcium chloride (Intravenous)	0.5 ml. of 5 per cent solution for each kilogram Repeat if necessary		
Calcium gluconate or lactate	1.25 Gm. (20 gr.)	2 Gm. (30 gr.)	Every 4 hours
Castor oil	4 ml. (1 dram)	8 ml. (2 drams)	One dose
Chloral hydrate (By enema)	0.2 Gm. (3 gr.)	0.3 Gm. (5 gr.)	P. r. n.*
Codeine sulfate	4 mg. ($\frac{1}{16}$ gr.)	5 mg. ($\frac{1}{12}$ gr.)	P. r. n.*
Coramine	See Nikethamide		
Diphenylhydantoin sodium (Dilantin)	15 mg. ($\frac{1}{4}$ gr.)	30 mg. ($\frac{1}{2}$ gr.)	2 to 3 times daily
Diuretin. See Theobromine sodiosalicylate			
Ephedrine sulfate	2.5 mg. ($\frac{1}{24}$ gr.)	4 mg. ($\frac{1}{16}$ gr.)	P. r. n.*
Epinephrin 1:1000 (Adrenalin)	0.125 ml. (2 min.)	0.2 ml. (3 min.)	P. r. n.*

*Pro re nata—whenever needed.

	6 MONTHS	1 YEAR	
Hyocine	See Scopolamine		
Iron ammonium citrate (maintenance)	40 mg. ($\frac{2}{3}$ gr.)	80 mg. (1.3 gr.)	Daily
Iron lactate	40 mg. ($\frac{2}{3}$ gr.)	80 mg. (1.3 gr.)	Daily
Magnesium sulfate (Intramuscular)	0.2 ml. 25% sol. for each kilogram		
Magnesia magma (Milk of Magnesia)	4 ml. (1 dram)	8 ml. (2 drams)	Daily
Methenamine (Urotropin)	0.3 Gm. (5 gr.)	0.5 Gm. ($7\frac{1}{2}$ gr.)	4 times daily
Morphine sulfate	1.5 mg. ($\frac{1}{48}$ gr.)	2 mg. ($\frac{1}{32}$ gr.)	P. r. n.*
Neostigmine methyl-sulfate injection 1:2,000 dilution	0.2 ml. (3 min.)	0.3 ml. (5 min.)	Every 4 to 6 hours
Nikethamide 25%	0.2 ml. (3 min.)	0.3 ml. (5 min.)	P. r. n.*
Paregoric	0.3 ml. (5 min.)	0.5 ml. (8 min.)	4 times daily
Penicillin (Intramuscular)	10,000 units	20,000 units	Every 3 hours
Phenobarbital	15 mg. ($\frac{1}{4}$ gr.)	30 mg. ($\frac{1}{2}$ gr.)	3 times daily
Posterior pituitary injection (Pituitrin (hypo))	0.125 ml. (2 min.)	0.2 ml. (3 min.)	P. r. n.*
Prostigmine	See Neostigmine		
Scopolamine hydrobromide (Hyocine)	0.15 mg. ($\frac{1}{480}$ gr.)	0.2 mg. ($\frac{1}{320}$ gr.)	P. r. n.*
Sodium bicarbonate (see table, page 484)	2 Gm. (30 gr.)	3 Gm. (45 gr.)	Every 4 hours
Sodium bromide	0.2 Gm. (3 gr.)	0.3 Gm. (5 gr.)	Every 4 hours
Sodium citrate	3 Gm. (40 gr.)	4 Gm. (60 gr.)	Every 4 hours
Sodium lactate	See table, page 484		
Streptomycin (Intramuscular)	45,000 units/kg./24 hours 20,000 units/pound/24 hours Divided into 8 doses		Every 3 hours
Sulfonamides	0.2 Gm. for each kilogram ($1\frac{1}{2}$ gr. for each pound) divided into 6 doses		Every 4 hours
Theobromine sodium-salicylate (Diuretin)	0.125 Gm. (2 gr.)	0.2 Gm. (3 gr.)	4 times daily
Thyroid	Start with 15 mg. ($\frac{1}{4}$ gr.) daily. Increase slowly to desired effect, up to 60 mg. (1 gr.) daily		
Tridione	0.2 Gm. (3 gr.)	0.3 Gm. (5 gr.)	3 to 4 times daily
Urotropin. See Methenamine			
Vitamin K (Therapeutic dosage)	For single administration 1 mg. For continued administration 0.1 mg. daily		

INDEX

A

- Absorption of carbohydrate, 59, 138
 - of fat, 139-140
 - of protein, 138
 - of water, 140
- Acetic acid for acidifying milk, 225
- Acetyl salicylic acid, 494
- Acetonuria in dysentery, 315
- Achlorhydria in celiac disease, 324
- Acid, acetic, for acidifying milk, 225
 - ascorbic, 122-126
 - boric, for ammonia dermatitis, 150
 - butyric, formed in colon, 144, 150, 288
 - in stomach, 278
 - in milk, 74
 - citric, for acidifying milk, 224
 - fatty, digestion of, 140
 - distribution in human and cow's milk, 158
 - folie, 19-20
 - hydrochloric, in treatment of tetany, 446
 - lactic, for acidifying milk, 220, 222-224
 - in blood in anhydremia, 284
 - mandelic for pyelitis, 404
 - nicotinic, 117
 - in milk, 158
 - organic, in blood of premature infant, 380
 - pantothenic, 110, 121
 - paraminobenzoic, 110, 121
 - phytic, in cereals, 414
 - production in diarrhea, 274
- Acid-ash foods, 94
- Acid-base metabolism, 79, 94
 - in diarrhea, 284-285
 - in premature infant, 380
 - in pyloric stenosis, 324
 - in vomiting, 353-355
- Acid-milk feedings, 218-228
- Acid-sugar solution, 228
- Acidity, gastric indigestion, 133, 134
 - (*see also* Digestion)
- Acidosis as cause of rickets, 417
 - of vomiting, 352
 - in anhydremia, 283, 284
 - in diarrhea, 79, 280, 284-285
 - in malnutrition, 258
 - in relief of tetany, 446
 - use of sodium lactate in, 300-301
 - water loss in, 80
- Activity, caloric allowance for, 47
- Adenoid infection as cause of otitis media, 397
 - of pyelitis, 405
 - in rickets, 424
- Adermin (pyridoxine), 120
- Adrenalin (epinephrin) as stimulant, 310, 494
- Aerobacter aerogenes in the intestine, 142, 143
 - in milk, 195
- Age, skeletal, determination of, 31
- Agglutinins in dysentery, 313
- "Air hunger" in acidosis, 283
- Air swallowing, as cause of colic, 356
 - of vomiting, 333
- Albumin milk, 235-236
- Albuminuria in newborn and immature infant, 380
 - in scurvy, 455
- Alecaligenes ammoniagenes as cause of ammonia dermatitis, 150
- Alimentary intoxication, 270-311 (*see also* Diarrhea)
- Alkali, administration of, in acidosis, 299, 300, 484
- Alkalosis as cause of tetany, 444
 - in diarrhea, 285
 - in pyloric stenosis, 341
 - in vomiting, 354-355
 - symptoms of, 354
 - treatment of, 355
- Allergy, 368-374
 - as cause of colic, 356, 357
 - of diarrhea, 277, 370
 - of eczema, 373
 - of vomiting, 352, 374
- Alpha-lobelin, use of, as respiratory stimulant, 386
- Amebiasis, 275
- Amines developed by bacteria in colon, 143
- Amino acids in metabolism, 52, 86, 118, 132, 138
 - in milk, 158
- Ammonia from bacterial action in intestine, 143
 - in urine, 150
- Ammoniacal diaper, 150
- Ammonium chloride, dosage of 355, 494
 - in treatment of alkalosis, 355
 - of pyelitis, 405
 - of tetany, 446
- Amylase in starch digestion, 139

- Amytal, sodium, 494
 Anal fissure in constipation, 363, 367
 Anaphylactic reactions in allergy, 368
 Anemia due to goat milk diet, 230
 to pyridoxine deficiency, 120
 folic acid in relation to, 119
 in malnutrition, 259
 in rickets, 424, 435
 in scurvy, 455, 458
 macrocytic, hyperchromic, 119, 230
 neonatorum, and vitamin K, 108
 nicotinic acid in relation to, 117
 nutritional, 90, 91
 of premature infant, 39, 88, 388, 392
 of sprue, 119
 Aneurin (*see* Thiamine)
 Anhydremia, 281-284, 298-311
 as cause of vomiting, 284, 352, 353, 391
 blood changes in, 282, 283
 fluid administration in, 485
 in mastoiditis, 398
 methenamine contraindicated in, 405
 red cell count in, 40
 secretion of urine in, 284
 Anomalies, congenital (*see* Malformations)
 Anorexia in celiac disease, 325
 in dysentery, 319
 Anoxemia in anhydremia, 284
 Anoxia of newborn, 376
 Anti-aerodynia vitamin (pyridoxine), 120
 Anti-beriberi vitamin (*see* Thiamine)
 Antidysentery sera, 320
 Antigen, definition of, 368
 Antineuritic vitamin (*see* Thiamine)
 Antipyrin, 494
 Antiseptics, use of, in diarrhea, 310
 Antisyphilitic treatment, 408
 Antivitamin, 118
 Antrotomy in treatment of mastoiditis, 401
 Antrum, mastoid, 397 (*see also* Mastoiditis)
 maxillary, 402 (*see also* Sinusitis)
 Apneic attacks in premature infant, 376, 385-386
 Apparatus for blood transfusion, 470-475
 for oxygen inhalation, 385-386
 for urine collection, 459
 Apple diet in diarrhea, 296
 in dysentery, 320
 Argyrol (*see* Silver protein)
 Arsenical drugs in syphilis, 408
 Artificial feeding, 182, 190 (*see also* Feeding, artificial; Milk, cow's)
 Artificially fed infant (*see also* Feeding, artificial)
 constipation in, 364
 cyanosis in, due to well water, 188
 incidence of diarrhea in, 272
 of rickets in, 413, 416
 of scurvy in, 450
 preference for various formulas, 190
 requirements of, mineral, 79, 83, 85, 88, 90, 91
 nutritional, 127-139
 vitamin, 100, 103, 112, 116, 117, 124
 water, 79
 stools of, 146
 Ascorbic acid, 122-126
 as supplement to breast-fed infant, 125, 177
 deficiency, 122, 450-458
 in formation of bone, 82
 in milk, cow's, 123, 158
 human, 124, 158
 in scurvy, 122, 450-458
 requirement of, 124, 239
 in early growth of embryo, 108
 in prematurity, 392
 sources of, 123, 124, 239
 synthetic, 122, 458
 Athrepsia, 251-269 (*see also* Malnutrition)
 Atony, intestinal, in rickets, 428
 Atopy, 368
 Atresia of duodenum, 349
 of esophagus, 339
 Atrophy, infantile, 251-269 (*see also* Malnutrition)
 Atropine sulfate, dosage of, 494
 use of, in colic, 360
 in diarrhea, 310
 in pyloric stenosis, 344
- B
- Bacillary dysentery, 312-322 (*see also* Dysentery)
 Bacteria, proteolytic, 196, 288
 Bacterial contamination of food, 187
 count of milk, 194, 196
 Bacterially soured milk in infant feeding, 221
 Bacteriology of gastrointestinal tract, 141-144
 of milk, 194-198
 of stools, 150

- Balsam of Peru ointment for chapped nipples, 165
 Banana in celiac disease, 68, 327, 328
 in infant feeding, 68, 246
 Barley gruel, 69
 Basal metabolism, 45-47
 Base, loss of, in diarrhea, 284
 Behavior, motor, of infant, 36-38
 Belladonna, tincture of, 494
 Benadryl, dosage of, 494
 Beriberi, 111 (*see also* Thiamine)
 Beta-alanine as gastric stimulant, 132
 Bichloride of mercury for ammoniacal diaper, 150
 Bile acids, 137
 in fat digestion, 137, 139
 pigment, stool color related to, 147
 salts, 139
 and vitamin K, 109
 Bilirubin, 147
 Biliverdin, 147
 Biotin, 120
 Bismuth, effect of, on color of stools, 148
 line in bone, 435
 salts in diarrhea, 309
 in dysentery, 320
 in syphilis, 408
 subcarbonate, dosage of, 310
 Blood, 38-41
 calcium in rickets, 423
 in tetany, 84, 440, 444
 normal, 84
 changes in anhydremia, 281, 283
 in dysentery, 315
 in malnutrition, 258, 259, 260
 circulation in anhydremia, 282
 in malnutrition, 258
 in newborn and premature infant, 377
 clotting and vitamin K, 106
 collection, technique, 462-468
 corpuscles, red, at birth, 38, 39
 in anhydremia, 282
 in malnutrition, 259
 white, at birth, 40
 differential count, 41
 grouping of, 468
 in dysentery, 315
 in malnutrition, 260
 hemoglobin (*see* Hemoglobin)
 in diarrhea, 282
 in dysentery, 315
 in infancy, 38-41
 in stools, 149
 in urine in scurvy, 455
 matching technique, 468
 phosphatase in rickets, 423
 phosphorus in rickets, 423
 Blood phosphorus—Cont'd
 in tetany, 440
 normal, 423
 pressure in infancy, 28
 prevention of clotting, 466, 470
 protein in celiac disease, 326
 in malnutrition, 260
 normal level, 260
 sugar, 59, 60
 transfusion apparatus, 470-474
 in treatment of anemia of rickets, 435
 of cardiac disease, 474
 of diarrhea, 304
 of malnutrition, 266-267
 of otitis media, 397
 of pneumonia, 474
 quantity of blood for, 474, 485
 technique of, 469-481
 vessels in vitamin C deficiency, 122, 454
 vitamin A, 99
 volume, 38
 in anhydremia, 281
 in malnutrition, 258
 Body, growth of, 17-42
 length of, in infancy, 20-22
 mineral composition of, 77
 proportions in infancy, 19
 temperature, normal, in infancy, 36
 of premature infants, 381-384
 water, 78-82
 weight, 22-24
 Boiling of milk for infant feeding, 188, 199, 210
 Bone, 30-31
 changes in rickets, 417-422, 424-428
 in scurvy, 451-454, 456
 deformities, congenital, 18
 ground glass appearance of, in scurvy, 452, 453
 in vitamin C deficiency (*see* Scurvy)
 marrow changes in rickets, 424
 in scurvy, 453
 transfusion, 478
 mineral content of, 31
 salts, formation of, 82
 Boric acid for ammoniacal diaper, 150
 Bosses in rickets, 425
 Bottle feeding (*see* Feeding, artificial)
 nursing, 211
 Bread in infant feeding, 246
 Breast feeding, amount of milk in, 169, 171
 contraindications to, 154-156, 410
 emotional satisfaction from, 153
 in prevention of diarrhea, 288
 of normal infant, 152-181

- Breast feeding—Cont'd
 of syphilitic infant, 410
 position of baby during, 170
 schedule of, 166-169
 technique of, 166-171
 milk, 156-161 (*see also* Milk, human)
 characteristics of, 156
 composition of, 156-161
 "dairies," 179
 manual expression of, 180-181
 unsuitable for feeding, 174
 pumps, 181
- Breast-fed infant, additions to diet of, 176-177
 characteristics of, 171-172
 constipation in, 365-366
 gastrointestinal disturbance of, 175
 mixed feedings for, 175
 nursing habits of, 168-169
 overfeeding of, 173
 stools of, 145
 undernutrition of, 172
 vitamin C requirements of, 124, 177
 vitamin D requirements of, 176
 vitamin deficiencies in, 153
 water requirement of, 80
 weaning of, 177
- Breasts, caked, 166
- Breck feeder for premature infant, 389
- Bronchitis complicating malnutrition, 259
 in relation to allergy, 370
- Brucella melitensis* in milk, 195
- Buffer value of milk, 193 (*see also* Milk)
- Buffered lactic acid solution, for intravenous use, 301
 for oral use, 299
 in treatment of diarrhea, 294, 299, 301, 303, 484
- Bulb feeding for premature infant, 389
- Buttermilk in infant feeding, 220
- Butyric acid, formation of, in gastrointestinal tract, 144, 150, 273, 288
 in milk, 74

C

- Caffein as stimulant, 310, 494
- Caffey's disease, 456
- Caked breasts, 166
- Calciferol (*see* Vitamin D)
- Calcium absorption, 83
 blood (*see* Blood calcium)

- Calcium—Cont'd
 chloride, dosage of, 494
 in treatment of tetany, 449
 content of body, 77, 82
 of premature infants, 83, 379
 of serum of normal infant, 84
 in rickets, 84, 423
 in tetany, 84, 440, 444
 gluconate, dosage of, 494
 in treatment of tetany, 446
 in cow's milk, 83, 158
 in human milk, 83, 158
 metabolism, 82-86, 431, 433
 in rickets, 415, 423
 salts in milk-free diets, 85
 in treatment of diarrhea, 311
 of tetany, 84, 445
 parenteral use of, 84, 445, 494
 unnecessary as addition to milk, 84
 in rickets, 435
 soaps, formation of, in intestine, 74, 140, 362
 urinary excretion of, 83
- Calomel, misuse of, 298
- Calorie value of condensed milk, 202, 233
 of cow's milk, 158, 191, 202
 of dried milk, 202, 230
 of evaporated milk, 202, 214
 of human milk, 157, 158
 of malted milk, 202, 233
 of skimmed milk, 202, 230
- Calories, allowance for activity, 47
 for growth, 47
 for unutilized food, 48
 requirement of, 48, 127
 in malnutrition, 263
 of premature infant, 379
 significance of, in metabolism, 43
- Cano sugar (sucrose), 59, 63, 70
- Capacity of stomach, 29
- Capillary changes in scurvy, 122, 454
- Carbohydrate for infant feeding, 58, 70
 in constipation, effect on stools, 362, 364
 metabolism, 58-70
 mixtures for infant feeding, 65-67
 of fruits, 68
 parenteral administration of, 70
 (*see also* Dextrose)
 requirement of, 58, 128
 storage of, 76
- Carbohydrate-milk mixtures for infant feeding, 234
- Carbon dioxide and oxygen inhalation for premature infants, 385

- Carbon dioxide—Cont'd
 of blood in pyloric stenosis, 342, 354
- Carboxylase, 111
- Carminatives in treatment of colic, 358
- Carotene (*see* Vitamin A)
- Carpopedal spasm, 354, 430, 437, 440, 442, 443
- Casein (*see also* Curds)
 hydrolysate as milk substitute, 237, 372
 in relation to allergy, 372
 of cow's milk, 158, 192
 of human milk, 157, 158
- Castor oil, 298, 494
- Cathartics, concentrated sugar solutions as, 70
 contraindicated in diarrhea, 297, 309
 in dysentery, 320
 in constipation, 366
- Celiac disease, 323-332
 absorption of vitamin A and, 99
 "celiac rickets," 326
- Cellular elements in stools, 149
- Cereal for thickened feedings, 337, 345
- Cereals in infant feeding, 243-245, 246
 in relation to allergy, 369
 to constipation, 365
 to rickets, 414
- Certified milk, 197
- Cevitamic acid (*see* Ascorbic acid)
- Chart, clinical, 490-493
 laboratory sheet for, 494
- Cheilitis due to riboflavin deficiency, 115
- Chemical preservatives in milk, 197
- Chemotherapy in dysentery, 321
- Chest deformity in rickets, 425, 426
 growth of, 27-28
- Chloral hydrate, dosage of, 494
- Chloride, loss, of, in pyloric stenosis, 341
 in vomiting, 355
 metabolism, of, 78-82
- Cholera infantum, 298-311 (*see* Anhydremia)
 and mastoiditis, 398
 and sinusitis, 402
- Cholesterol, irradiated, 103 (*see also* Vitamin D)
- Choline, 121
- Chronic intestinal indigestion, 323 (*see also* Celiac disease)
- Chvostek's sign, 354, 443
- Chyme, 137
- Circulation (*see also* Blood)
 in newborn and premature, 377
- Citrated blood for transfusion, 469
- Citric acid milk, 224
- Citrin, 126
- Citrus fruits as sources of vitamin C, 123, 239
- Clinical chart, 490-493
- Clothing of premature infant, 381
- Clotting of blood and vitamin K, 107
- Cobalt, 93
- Coccarboxylase, 112
- Codeine sulfate, dosage of, 494
- Cod-liver oil (*see also* Vitamin D)
 as source of iodine, 92
 requirement for breast-fed infant, 176
 for normal infant, 104, 105, 176, 238
 for premature infant, 391
 in rickets, 434
 time of addition of, 105
- Coenzyme, 112
- Cold, common, treatment of, 393
- Colic, 356-360
 allergy in relation to, 370
- Colitis, membranes, 149
- Colon bacillus (*see* Escherichia coli)
- Color of stools, 147-148
- Colostrum, 156
 vitamin A of, 98
- Complemental feedings, 175
- Condensed milk, 232
- Congenital anomalies (*see* Malformations)
- Constipation, 361-367
 fruits and vegetables in treatment of, 249, 365
 in pellagra, 118
 in rickets, 428
 relation of carbohydrate intake to, 70
 of fat intake to, 74, 257
 of protein intake to, 56, 257, 362
- Constitutional weakness, cause of malnutrition, 255
- Contraindications to breast feedings, 154
- Convulsions, 449
 in dysentery, 314
 in hypoglycemia, 60
 in pyloric stenosis, 342
 in severe vomiting, 354
 in tetany, 342, 437, 442, 444
 therapy, 449
- Copper, metabolism of, 90
- Coramine, dosage of, 494
- Corn syrup, 66

- Corynebacterium diphtheriae as milk contaminant, 195
 Costochondral junctions, enlargement of, in rickets, 425, 426
 Cow's milk (*see* Milk, cow's)
 Creatinine as measure of basal rate, 46
 Cretinism, congenital, 41
 Cultures, stool, technique of, 461
 urine, technique of, 461
 Curds in cow's milk, 133, 134, 184
 (*see also* Milk, cow's)
 in human milk, 133
 in stools, 148
 Custard, soft, 241
 Cyanosis from well water, 188
 in diarrhea, 311
 in premature infants, 385
 Cyclic vomiting, 352
 Cystic disease, absorption of vitamin A in, 99
 fibrosis of the pancreas, 330-332
 Cystine, 52, 158

D

- Dark adaptation and vitamin A, 97, 100, 101
 Darrow's buffered potassium chloride solution, 299, 302-303, 484
 formula for, 303
 treatment scheme for parenteral use, 484
 Deformities caused by rickets, 424-430
 correction of, 435-436
 Dehydration in malnutrition, 261, 268
 in otitis media, 397
 in premature infants, 391
 in pyloric stenosis, 342
 7-Dehydrocholesterol, irradiated (vitamin D₃), 103
 "Dekomposition" (*see* Malnutrition)
 Dental caries, effect of fluorine on, 92
 Dentition, 26
 and vitamin D, 104
 delayed, in rickets, 430
 Development, 17-42 (*see also* Growth)
 of motor behavior, 36
 of nervous system, 33
 of premature infant, 375, 392
 of special senses, 35
 Dextrin, 59, 64, 69, 70
 Dextrin-maltose mixtures, 61, 65
 with added potassium bicarbonate, 66
 vitamin B-complex, 65
 Dextrin-maltose-dextrose mixtures, 66
 Dextrin-maltose-dextrose-sucrose mixtures, 66
 Dextrose, content of blood, 59, 60
 in celiac diet, 328
 in diarrhea, 306
 in infant diet, 59, 61, 64, 65, 69
 intravenous administration of, in diarrhea, 293, 302
 in malnutrition, 267
 rate of, 486
 technique of, 483-484
 Diabetes as cause of malnutrition, 256
 Diarrhea, 270-311 (*see also* Dysentery)
 absorption in, 99, 280
 acidosis associated with, 284-285
 allergy as cause of, 277, 370
 anhydremia in, 281-284
 as cause of death, 270
 bacteria as cause of, 259, 272-274
 butyric acid and, 144, 273
 cathartics in, 297, 309
 causes of, 270-280
 differential diagnosis of, 285-288
 drugs in treatment of, 309-311
 dysentery organisms in relation to, 275, 312
 effects of, on body, 280-285
 enteral infections as cause of, 271, 287
 epidemic, of newborn, 276
 fatty acids as cause of, 74
 feedings during, 306
 in malnutrition, 259, 261, 265
 in mastoiditis, 398, 399
 in otitis media, 394
 in pellagra, 118
 in premature infants, 390-391
 in pyelitis, 403
 in pyloric stenosis, 341
 in relation to fat intake, 74
 in sinusitis, 402-403
 parenteral infections as cause of, 268, 271, 286, 295, 394, 398, 400
 prophylaxis of, 288-290
 severe, treatment of, 298-309
 starvation, as cause of, 252, 261, 277, 286
 stools in, 146
 sugars as cause of, 69
 symptoms of, 278-280, 298
 treatment, 290-309
 feedings in, 292, 306
 in artificially fed infants, 295-309
 in breast-fed infants, 293-295
 medicinal, 309-311

- Diarrhea, treatment—Cont'd
 of mild type, 295-298
 of severe type, 298-309
 virus as cause of, 276
 Diet as cause of rickets, 379, 413
 (*see also* Feedings, Foods)
 of scurvy, 450
 elimination, for allergy, 373
 for second year, 248-250
 in treatment of celiac disease, 326-330
 of diarrhea, 291-292, 296-297, 305-306
 of dysentery, 318-320
 of pyloric stenosis, 345-347
 ketogenic, 76
 of normal infant, 238-250
 Difficult feeding cases, 253
 Digestion, 131-144
 Digestive capacity of breast-fed infant, 29
 of premature infant, 378
 Dihydrotachysterol as an antirachitic, 103
 in treatment of tetany, 448
 Diphenylhydantoin sodium (Dilantin), dosage of, 494
 Diphtheria, bacilli in milk, 195
 Diuretin, dosage of, 494
 Dried milk, 83, 202, 230-232 (*see also* Milk, cow's)
 Drugs, dosage, table of, 494-495
 and allergy, 369
 excretion of, in human milk, 165
 in treatment of constipation, 366, 367
 of diarrhea, 309
 of dysentery, 320-322
 of pyloric stenosis, 344, 345, 347
 Duodenum, obstruction of, 349
 Dysentery, bacillary, 141, 149, 151, 275, 278, 312-322
 chemotherapy in, 321-322
 complications of, 316-317
 pathology of, 312
 symptoms of, 314-316
 treatment of, 318

E

- E. typhosa, as cause of diarrhea, 275
 in milk, 195
 Ear infections (*see* Otitis media)
 Ecchymosis in scurvy, 455
 Eczema in relation to allergy, 373
 to unsaturated fatty acids, 73
 Edema in celiac disease, 326
 in malnutrition, 55, 260
 Eggs in infant diet, 240-242
 sensitization to, 240, 369

- Eiweiss Milch, 235, 236
 Elimination diets for allergy, 373, 374
 Endocrine disturbances as cause of malnutrition, 256
 of rickets, 417
 of tetany, 437
 Enemas in constipation, 366
 retention of, in dysentery, 320
 Energy metabolism, 43-51 (*see also* Calories)
 requirements of infants, 48-51, 127
 of premature infant, 379
 Enteral infections as cause of diarrhea, 287 (*see also* Dysentery)
 Enterokinase, 137
 Enzymes, digestive, 131-133, 137-140
 of milk, 193
 oxidation-reduction, 110, 111, 115, 116
 respiratory, 110, 111, 115, 116
 yellow, of Warburg, 110, 115
 Ephedrine, dosage of, 494
 in treatment of otitis media, 396
 Epinephrin as stimulant, 310
 dosage of, 494
 Epiphyseal changes in rickets, 417-422
 in scurvy, 451, 452, 458
 Epithelial changes in riboflavin deficiency, 115
 in vitamin A deficiency, 96
 Erb's sign in tetany, 443
 Ergosterol, irradiated (*see* Vitamin D)
 Erythroblastosis fetalis, transfusion in, 469
 Erythrocytes (*see* Blood corpuscles)
 Escherichia coli as cause of diarrhea, 273, 287
 of mastoiditis, 400
 of pyelitis, 403
 in duodenum, 141, 273
 in milk, 195
 in normal intestine, 141, 142
 in urine cultures, 461
 Evaporated milk, composition of, 202, 213-215
 in formula, 215
 in acid milk, 227
 vitamins in, 113, 123
 Exsiccosis, 281
 Extracellular water, 79
 Extremities, deformities of, in rickets, 426-428
 tenderness of, in scurvy, 454
 Exudative diathesis, in relation to allergy, 370
 Eyes, congenital deformities of, 18, 19, 116

F

- Face in infancy, 25
- Facial phenomenon in tetany (Chvostek's sign), 443
- Fat as carrier of vitamins, 73, 95
 composition of, 71, 73, 158
 digestion of, in intestine, 137, 139-140
 in stomach, 135
 in food of premature infant, 389
 in ketogenic diet, 76
 metabolism of, 71-76
 of cow's milk, 73-75, 158, 191
 of human milk, 73-74, 157, 158
 parenteral administration of, 75
 requirement of, 128
 tolerance of, in celiac disease, 329
- Fatty acids essential, 74
 unsaturated, in milk, 158
 in eczema, 72
- Feces (*see* Stools)
- Feeding, artificial, 182-189
 acid milk mixtures, 218-228
 in diarrhea, 292, 306
 in dysentery, 318-320
 carbohydrates in, 58-70, 232 (*see also* Carbohydrate)
 complementary, 175
 condensed milk in, 232-233
 construction of formulas, for, 205-209, 215-217, 225-227
 diluents for, 203
 dried milk in, 230-232
 evaporated milk mixtures in, 213-217, 225-227
 foods, other than milk in, 237
 formulas, construction of, acid-milk, 225
 digestibility of, 188-190
 dried milk, 231
 evaporated milk, 215, 227
 sweet milk, 205
 fruit in, 68, 243, 246
 goat's milk in, 229
 malted milk in, 233-234
 number of, 205
 of premature infant, 388-389
 parenteral (*see* Intravenous therapy)
 percentage method of calculating, 183
 preference of infant for, 190
 preparation of, 209
 proprietary foods in, 229-237
 protein milk in, 235
 requirements for satisfactory, 185-187
 rickets in relation to, 413
- Feeding, artificial—Cont'd
 skimmed milk, in diarrhea, 292, 306
 in dysentery, 318, 319
 special foods in, 229-237
 supplemental, 175
 sweet milk mixtures, 205-209
 technique of feeding, 212
 of preparing, 209-211
 thickened, in pyloric stenosis, 345
 in treatment of rumination, 337
 volume of, 204
 breast (*see* Breast feeding)
 mixed, 175
- Ferric ammonium citrate (*see* Iron)
- Ferrous sulfate (*see* Iron)
- Fetus, nutrition of, 18
- Fever in infants with anhydremia, 283, 297
 with dysentery, 314
 with mastoiditis, 399
 with otitis media, 395
 with rhinopharyngitis, 394
- Flat feet as cause of knock-knee, 428
- Flatulence (*see* Colic)
- Flexner bacillus as cause of dysentery, 312
- Flügge, bacillus of, 141, 276
- Fluid administration, oral, in anhydremia, 299
 parenteral, 484-485
 in anhydremia, 300-303
 in malnutrition, 267-268
- Fluorine, metabolism of, 92-93
- Folic acid, 119
- Foods (*see also* Feeding, artificial)
 bacterial contamination of, 187
 for special conditions, 235-237
 proprietary, 229-237
 requirement of underweight infant, 50
 unsuitable composition of, as cause of vomiting, 335
- Formulas, milk (*see also* Feeding, artificial)
 acid, 226
 evaporated, 216
 sweet, 209
- Fractures in rickets, 419
 in scurvy, 453
- Fredet-Rammstedt operation for pyloric stenosis, 348
- Fruits, carbohydrates of, 68
 in infant feeding, 68, 243, 246
 in relation to constipation, 249, 365

G

- Galactose, 59
 Gamma, a measure of weight, 112
 Gas bacillus (*see* *Clostridium perfringens*)
 Gastric achlorhydria in celiac disease, 324
 capacity, 29
 digestion, 132-137
 juice, alkalosis due to loss of, 341, 354
 concentration of, 132, 378
 in malnutrition, 264
 lavago in pyloric stenosis, 347
 Gastroenteritis (*see* *Diarrhea*)
 Gastroenterospasm, 357
 as cause of colic, 357
 of diarrhea, 277, 286
 of vomiting, 338
 in relation to allergy, 370
 treatment of colic caused by, 360
 Gastrointestinal changes in rickets, 428
 disturbances from iron salts, 90
 in relation to fat and protein intake, 74
 to mastoiditis, 398
 to otitis media, 394
 to pyelitis, 403
 to sinusitis, 402, 403
 of breast-fed infants, 175
 tract, bacteriology of, 141-144
 irritation of, 271
 Gavage, of premature infant, 389
 technique of, 487-488
 Gee-Herter Disease (*see* *Celiac disease*)
 Gee's Disease (*see* *Celiac disease*)
 Gelatin, an incomplete protein, 52
 as milk modifier, 56
 Giardia lamblia, 323
 Glossitis due to riboflavin deficiency, 115
 Glucose (*see* *Dextrose*)
 tolerance curves in celiac disease, 326
 Glycerin suppositories, 366
 Glycerol, component as part of fat, 71
 Glycogen, 60
 Goat's milk, 229, 312
 Goiter (*see* *Thyroid*)
 Grasping reflex in newborn, 34
 Gray powder in treatment of syphilis, 408
 Growth, and development, 17-42
 and rickets, 415-416
 body proportions during, 19

Growth—Cont'd

- caloric allowance for, 47
 effect of vitamin D on, 104-105
 endocrines and, 17
 in celiac disease, 326
 in weight, 22-24
 linear, 20-22
 nutrition and, 18
 of endocrine glands, 41
 of premature infants, 24, 379, 392
 of skeleton, 30-31
 vitamin A and, 96
 vitamin D and, 104
 Gums in scurvy, 454

H

- Habit, in relation to constipation, 363, 366
 to eating, 249
 to vomiting, 337
 Hair, 32
 Harrison's groove in rickets, 426
 Hartmann's buffered lactic acid solution, 268, 293, 301
 for oral use, 299
 technique of administration of, 484-485
 Head, circumference of, 25
 growth of, 24
 in rickets, 425
 Hearing, sense of, 35
 Heart, block, from potassium intoxication, 304
 changes in malnutrition, 260
 in scurvy, 455
 congenital anomalies of, 19
 and nutrition, 256
 development, 28
 growth of, in premature infants, 377
 Heat regulation in newborn and premature, 378
 Hematuria from methenamine therapy, 405
 in scurvy, 455
 Hemoglobin (*see also* *Anemia*)
 in infancy, 38, 87
 in premature infants, 39, 88
 Hemogram, Schilling, 41
 in dysentery, 315
 in malnutrition, 260
 Hemolytic streptococci, in milk, cow's, 195
 Hemorrhage, in dysentery, 313, 314
 in premature infants, 377
 in purpura, 457
 in scurvy, 122, 450, 453, 454, 455
 Hemorrhagic disease of newborn, 108

Hernia, umbilical, in rickets, 428
 Hesperidin, 126
 Hiss-Russell bacillus, 312
 Histamine in relation to diarrhea, 143, 274, 288
 History taking, outline for, 488-490
 Holstein milk, 191
 Homogenization of milk, 75, 200
 Honey in infant feeding, 68
 Human milk (*see* Milk, human)
 Humidity, control of, for premature infants, 383
 Hunger diarrhea, 277
 Hydrobilirubin, 147
 Hydrochloric acid, in treatment of tetany, 446
 milk, in treatment of tetany, 446
 of gastric juice, 132, 264, 378
 Hydrolability in diarrhea, 143, 274, 281
 Hydrolyzed casein in allergy, 372
 Hygeia nipples for thick feedings, 337
 Hyocine, 495
 Hypertonic dextrose solution in diarrhea, 293, 305
 in malnutrition, 267
 infant, 357
 Hypoglycemia in newborn, 60
 Hypoparathyroidism, cause of tetany, 437
 Hypothrepsia (*see* Malnutrition)
 Hypothyroidism, cause of constipation, 361
 growth retardation, 256
 recovery from, associated with rickets, 415

I

Idiopathic steatorrhea (*see* Celiac disease)
 Ileocolitis, 312-322
 Incubator bed for premature infants, 384
 rooms for premature infants, 383
 Indicator for blood flow in transfusion, 474
 Indigestion, chronic, intestinal (*see* Celiac disease)
 Indol developed by bacteria, 143
 Infant foods, proprietary, 232, 234, 237
 Infantile cortical hyperostoses, 456-457
 Infantilism, intestinal (*see* Celiac disease)
 Infections, as cause of diarrhea, 268, 271, 272, 276, 286, 287, 295, 298, 394, 398, 400, 402, 403
 of malnutrition, 254-255
 of vomiting, 336
 ascorbic acid requirement in, 126
 associated with nutritional disturbances, 131, 393-410
 in premature infants, 384
 secondary to diarrhea, 308-309
 to malnutrition, 259
 to rickets, 424
 Infectious diarrhea (*see* Dysentery)
 Inositol, 121
 Insulin in newborn, 60
 use of, in malnutrition, 61
 Interstitial keratitis, due to riboflavin deficiency, 115
 water, 79
 Intestinal atony in rickets, 428
 digestion, 137-141
 enzymes, 137
 indigestion, chronic (*see* Celiac disease)
 infantilism (*see* Celiac disease)
 obstruction, vomiting in, 349-351
 tract, growth of, 28-29
 Intestine, bacterial action in, 139
 development of, 29
 Intoxication, alimentary, 298-311
 Intracellular water, 78
 Intracranial birth injury as cause of malnutrition, 256
 hemorrhage and vitamin K, 109
 in premature infants, 276
 Intraperitoneal administration of fluids, 482-483
 Intravenous therapy, continuous, 484-485 (*see also* Dextrose, Ringer's solution, Transfusion)
 in treatment of diarrhea, 293, 300-302
 of malnutrition, 267-268
 technique of, 469-487
 Intussusception as cause of vomiting, 350-352
 Iodine, 91-92
 Iron, 87-90
 and ammonium citrate, 90, 495
 in enzyme systems, 117
 lactate, 495
 medication in scurvy, 458
 requirement, of infant, 87-90
 of premature infant, 392
 reserves at birth, 39, 87
 salts, utilization of, 89-90
 stool color related to, 148

Irradiated cholesterol (*see* Vitamin D)
 ergosterol (*see* Vitamin D)
 Irrigation of colon in treatment of dysentery, 320
 Irritability, neuromuscular, in rickets, 430
 Islands of Langerhans, 41
 Isotonic sodium chloride (*see* Physiological salt solution)

J

Jacket for premature infant, 381-382
 von Jaksch's anemia accompanying rickets, 424
 Jaundice, physiologic, 39
 Jersey milk, 191
 Juice, fruit, in infant feeding, 246 (*see also* Fruit; Orange juice)
 gastric, 132, 264, 378
 pancreatic, 137
 succus entericus, 137

K

Keratitis, interstitial, due to riboflavin deficiency, 115
 Kernig sign in tetany, 442
 Ketogenic diet as dehydrating agent, 76
 Ketone bodies, formation of, 72
 Ketosis in immature infant, 72
 in malnutrition, 258
 Kidney, development of, 30
 dysfunction, cause of rickets, 417
 of premature infant, 380
 Knock-knee in rickets, 426, 428, 429
 Kyphosis in rickets, 428

L

Laboratory sheet for clinical chart, 492, 493, 494
 Laetalbumin in milk, 53, 158
 in relation to allergy, 369
 Lactate, buffered, 299, 301
 Lactation, diet during, 162
 hygiene during, 161
 in infant, 42
 Lactic acid milk, 220-224 (*see also* Feeding, artificial)
 dried, 223
 skimmed, 223
 producing organisms in milk, 195, 221
 solution, buffered, formula for, 299, 301

Lactic acid solution—Cont'd
 in diarrhea, 294, 299, 301, 303
 Lactobacillus acidophilus for souring milk for infant feeding, 221
 in intestine, 141-144
 in milk, 195
 bifidus in gastrointestinal tract, 141-143
 bulgaricus in milk, 195
 Lactoflavin (riboflavin), 115-116
 Lactose in infant feeding, 59, 62, 70
 Lanolin, use of, for chapped nipples, 165
 Lanugo of newborn, 32
 Laryngospasm in rachitic tetany, 430
 in tetany, 430, 437, 442, 444
 Latent tetany, 440, 441
 Laudanum in dysentery, 320
 Lavage, gastric, in pyloric stenosis, 347
 Laxative, drugs, dosage of, 494-495 (*see also* Cathartics)
 effect of colostrum, 156
 of molasses, 67
 of sugars, 62, 66, 67, 69
 Length, body, growth in, 20-22
 Leucocytes (*see* Blood corpuseles, white)
 Leukocytosis in dysentery, 315
 Leukopenia in dysentery, 315
 Ligaments, lax, in rickets, 424, 428
 Line, scurvy, in bone, 453
 Lipase, 135, 139
 Liver, growth of, 29-30
 Luminal (*see* Phenobarbital), 495
 Lungs, growth of, 28
 Lymphatic system, 32-33
 Lymphocytosis in rickets, 424

M

M. ovalis (*see* Streptococcus fecalis)
 M. tuberculosis in milk, 195, 406
 Magnesia, milk of, dosage, 495
 in treatment of constipation, 367
 Magnesium, 77, 86
 sulfate, dosage of, 495
 use of, in tetany, 86, 447, 449
 Malformations, as cause of malnutrition, 255-256
 atresias of gastrointestinal tract, 339, 349
 congenital, 18
 obliteration of bile duct and vitamin K, 109
 Malnutrition, 251-269
 as cause of celiac disease, 324
 blood changes in, 260

Malnutrition—Cont'd

- causes of, 251-261
- diarrhea in, 259, 261, 265
- food requirement in, 263
- hydrolability in, 260, 268
- in pyloric stenosis, 344, 348
- infections complicating, 259, 268
- intravenous therapy in, 266-268
- pathology of, 257-261
- Malt soup extract, 66
 - in treatment of constipation, 364, 365
- Malted milk in infant feeding, 61, 233
- Maltose in infant feeding, 65
 - utilization of intravenous, 59
- Maltose-dextrin mixtures in infant feeding, 61, 65
- Mandelic acid in pyelitis, 404
- Manganese, metabolism of, 93
- Manual expression of breast milk, 180-181
- Marasmus (*see* Malnutrition)
- Mastitis, contraindication to breast feeding, 155
- Mastoiditis, 397-402
 - as cause of diarrhea, 308
 - complicating malnutrition, 255
- Maxillary sinusitis (*see* Sinusitis)
- Meat in infant's diet, 245-246
- Meatal ulcer from ammoniacal diaper, 150
- Mechanical expression of breast milk, 180-181
- Meconium, 145
- Mehlnährschaden, 253, 257
- Menadione, 107
- Menstruation not contraindication to breast feeding, 155
- Mental development in rickets, 431
 - measured by motor behavior, 37-38
- Mercury in treatment of syphilis, 408
- Metabolism, acid base, 94
 - basal, 45-47
 - carbohydrate, 58-70
 - energy, 43-51
 - fat, 71-76
 - mineral, 77-104
 - protein, 52-57
 - vitamin, 95-126
 - water, 78-82
- Metaphyseal changes in rickets, 417, 419-422, 431
 - in scurvy, 451-453
- Methemoglobin, in well-water cyanosis, 188
- Methenamine, dosage of, 495
 - in treatment of pyelitis, 404
- Methionine in protein, 52, 158, 159
- Microgram, definition of, 112

- Microorganisms, growth of, in intestinal tract, 141-144
- Milch, Eiweiss, 235
- Milchnährschaden, 257
- Milk, cow's (*see* Feeding, artificial)
 - ascorbic acid content of, 123, 124, 158
 - bacteriology of, 194-197
 - boiled, 199
 - buffer value of, 134, 193
 - calcium of, 83, 158
 - carbohydrate mixtures, 234-235
 - containing corn oil, 235
 - simulating human milk, 235
 - casein curds from, in stomach, 133
 - certified, 197-198
 - chemical preservatives in, 197
 - composition and character of, 158, 191-202
 - condensed, sweetened, 61, 232
 - copper of, 90, 158
 - digestibility of formulas, 188
 - dried, 83, 202, 230-232
 - "Eiweiss," 235-236
 - enzymes of, 193
 - evaporated, characteristics of, 202, 213-215
 - fortified with vitamin D, 201, 215
 - loss of vitamins in, 214
 - fat of, 158, 202, 214
 - fatty acids of, 158
 - feedings (*see* Feeding, artificial)
 - formulas, bacterial contamination of, 187
 - homogenized, 75, 200
 - hypoallergenic, 236
 - in diet of normal infant, 238
 - iodine of, 91, 92
 - iron of, 89, 158
 - malted, in infant feeding, 61, 233-234
 - minerals of, 158
 - miscellaneous constituents of, 193
 - modification of, early attempts at, 182-184
 - for infant feeding, 201-202
 - for premature infant, 388-389
 - nicotinic acid content of, 117, 158
 - pasteurization of, 197, 198-199
 - loss of vitamins in, 113, 123, 199
 - phosphorus of, 83, 158
 - physical properties of, 184
 - "protein," 235-236
 - protein of, 53, 158, 192
 - rennin curds of, 133, 184
 - riboflavin content of, 116, 158

Milk, evaporated—Cont'd
 soft curd, 199-201
 substitutes in infant feeding, 237
 sulfur of, 158
 sweet, mixtures, 203-212
 thiamine of, 113-114, 158
 vitamin A content of, 98, 158
 with vitamin D added, 104, 201, 215

Milk, goat's 229-230
 in allergy, 372

Milk, human, allergy to, 369
 amino acids in, 158, 159
 amount secreted, 161-162
 received by infant, 169, 171
 bacteria of, 160
 casein curds from, 133
 characteristics of, 156-161
 colostrum, 156
 composition of, 156-158
 copper of, 90, 158
 deficiency of, 153
 drugs secreted in, 165
 expression of, 180-181
 fat in, 157, 158
 fatty acids in, 158
 fortification of, for prematures, 83, 388
 frozen, for infant feeding, 180
 immune bodies in, 152
 in feeding of premature infant, 387-388
 iodine of, 92, 158
 iron of, 89, 158
 lactalbumin of, 157, 158, 159
 minerals, 158, 159
 nicotinic acid of, 117, 158
 protein of, 157, 158, 159
 requirement of, by infant, 171
 riboflavin of, 116, 158
 rickets and, 413
 sugar in, 158, 159
 thiamine of, 113, 158
 unsuitable for infant, 174
 vitamin A of, 98, 158
 vitamin C of, 124, 158

Milk of magnesia, dosage of, 495
 in treatment of constipation, 367

Milk sickness in cows, 193

Milk sugar (*see* Lactose)

Milk-free foods in infant feeding, 237, 372

Mineral content of body, 77
 oil, contraindicated, 98, 99, 366
 salts, requirement of, 129
 skeletal, 82

Minerals, effect of illness on retention of, 84
 in milk, 158
 metabolism of, 77-94
 requirements, 129
 in malnutrition, 263
 in prematurity, 388

"Minimum perceptible" light test, 101

Mixed feeding, 175
 sugars in infant feeding, 59, 67

Molasses, 67

Monosaccharides, 59

Morphine sulfate, dosage of, 495

Mother, nursing (*see* Nursing mother)

Motor behavior of normal infants, 37-38
 of rachitic infants, 426, 428, 430

Mottled enamel, 93

Mucus in stools, 149

Murphy drip bulb, use in transfusion apparatus, 474

Muscle, effect of vitamin D on, 104
 growth of, 36
 in rickets, 424
 intestinal, atonic and hypertonic, 147
 weakness in celiac disease, 323, 326

Myxedema, 41

N

Napthoquinone, 107 (*see also* Vitamin K)

Negro, rickets in, 411-412

Neostigmine methyl-sulfate, dosage of, 495

Nephritis, chronic, in relation to rickets, 417
 in relation to tetany, 444, 449

Nervous system, development of, 33
 vomiting, 337

Neuropathic diathesis, 357, 370

Newborn period, the, 375-381
 tetany of, 437, 449

Niacin (*see* Nicotinic acid)

Nicotinic acid, 117
 in milk, 158

Night blindness in vitamin A deficiency, 100, 101

Nikethamide, dosage of, 495

Nipple shields, 165

Nipples, care of mother's, 165
 of rubber, 211
 for use with thick feedings, 337

Nitrates in well water, cause of cyanosis, 188

Nitrites in well water, cause of cyanosis, 188
 methemoglobin from, 188
 Normal saline, 484, 485
 Nursing bottle, 211
 mother, care of breasts of, 165-166
 diet of, 162-163
 hygiene of, 161
 position of baby during, 170, 212
 Nutrition, effect on growth, 17 (*see also* Feeding, Malnutrition)
 overnutrition, protein, 55
 Nutritional disturbances (*see* Malnutrition)
 requirements of infants, summary, 127-130

O

Ointments for chapped nipples, 165
 Opium (*see* Paregoric)
 Orange juice, ascorbic acid in, 239
 in diet, in celiac disease, 328
 in dysentery, 319
 of breast-fed infants, 177
 of normal infant, 239
 of premature infants, 392
 in treatment of scurvy, 457-458
 Organic acids, as cause of diarrhea, 272-273
 in blood of premature infant, 380
 Osteoblastic activity in scurvy, 451
 Osteochondritis of syphilis, 456
 Osteoclastic activity in scurvy, 451
 Osteomalacia, 415
 Osteoporosis in celiac disease, 326
 Osteotomy for rachitic deformities, 436
 Otitis media, 394-397
 as cause of diarrhea, 271, 286, 295, 308
 of vomiting, 336
 as complication in dysentery, 316
 in malnutrition, 259, 269
 in pyloric stenosis, 342
 in vomiting, 342
 mastoid infection in, 397
 treatment of, 396
 Ova, pinworm, 462
 Overdistention of gastrointestinal tract, cause of colic, 356
 Overfeeding as cause of diarrhea, 287
 Overnutrition, protein, 55
 Overweight infants, food requirements for, 127
 Oxalate, to prevent blood clotting, 466

Oxygen administration for premature infant, 385-386
 as treatment of diarrhea, 311
 in toxemia, 311
 technique of, 385-386

P

Pain, sense of, 35
 as symptom of scurvy, 455
 Pancreatic fibrosis, 330-332
 insufficiency, 330-332
 juice, 137 (*see also* Digestion)
 Pantothenic acid, 110, 121
 Paracentesis in otitis media, 396
 Paratyphoid bacillus as cause of dysentery, 275, 312
 of mastoiditis, 400
 Paraminobenzoic acid, 110, 121
 Parathyroid extract in treatment of tetany, 448
 gland, and bone metabolism, 82
 dysfunction and rickets, 417
 in tetany, 41, 437
 Paratyphoid bacillus as cause of mastoiditis, 400
 in intestinal tract, 141
 in milk, 195
 in stool cultures, 462
 Paregoric, dosage of, 495
 in treatment of colic, 358
 of diarrhea, 309
 of dysentery, 320
 Parenteral administration, 482-483, 484-485
 of calcium salts in tetany, 446
 of carbohydrate (*see* Dextrose)
 of electrolytes, 484, 485
 of magnesium salts in tetany, 447, 448, 449
 infections (*see* Infections)
 Pasteurization of milk, 197, 198-199
 Pathogenic organisms in cow's milk, 195
 Pectin in diarrhea, 296
 in dysentery, 320
 Pectin-agar-dextrin-maltose in diarrhea, 296
 Pellagra, 117, 118
 Penicillin, dosage of, 495
 in treatment of syphilis, 408
 Pepsin in gastric digestion, 132
 Percentage system of infant feeding, 183
 Peristalsis, gastric, visible in pyloric stenosis, 341
 Peritoneal administration (*see* Intraperitoneal administration)
 Petrolatum in care of breasts, 165

- Phenobarbital, dosage of, 495
 in colic, 358
 in diarrhea, 310
 in dysentery, 320
 in pyloric stenosis, 344, 347
- Phenol in glycerin in treatment of
 otitis media, 396
- Phosphatase, serum, in Caffey's dis-
 ease, 457
 in rickets, 423
 in tetany, 444
- Phosphorus in body, 77, 82-86
 content of cow's milk, 85, 158
 of human milk, 85, 158
 elementary, in treatment of rick-
 ets, 435
 of blood in rickets, 423
 in tetany, 440, 444
- Photophobia in riboflavin deficiency,
 115
- Physical examination, outline for, 489
- Physiological buffer salt solution (*see*
also Intravenous therapy)
 for intravenous therapy, 293,
 301, 484, 485
 for oral use, 299
 salt solution for intravenous ther-
 apy, 484, 485
- Phytic acid and rickets, 414
- Pituitrin, dosage of, 495
 effect on milk flow, 173
- Plantar reflex in newborn, 34
- Pneumococci in urine cultures, 461
- Pneumonia complicating dysentery,
 316-317
 lipoid, in premature infant, 391
- Porphyria, 117
- Posterior pituitary, dosage of, 495
 (*see also* Pituitrin)
- Posture in rickets, 426-430
- Potassium, 78
 carbonate added to sugar mixtures
 as laxative, 66
 in plasma, 303
 intoxication, 304
- Pregnancy, breast feeding during, 155
- Prematurely born infants, 375-392
 and newborn period, 375-381
 anemia in, 88, 392
 calcification of skeleton of, 379
 characteristics of, 375
 circulation in, 377
 cyanosis of, therapy of, 385
 development of, 24, 392
 digestive system of, 378
 fat absorption by, 102, 235
 feeding of, 386-390
 growth of, 24, 379, 392
 heat regulation of, 378
- Prematurely born infants—Cont'd
 incidence of, 18
 management of, 381-392
 nutritional requirements of,
 379
 organic acids in blood of, 380-
 381
 prothrombin, blood, in, 108
 respiration of, 375-377, 385
 rickets in, 379, 415, 433
- Preservatives, use of, in milk, 197
- Projectile vomiting in otitis media,
 394
 in pyloric stenosis, 342
- Prolapse of rectum in dysentery, 317
- Proprietary foods in infant feeding,
 232-237
- Prostigmine, dosage of, 495
- Protein, composition of, 52
 digestion of, 133, 138
 in diet of celiac disease, 327-328
 metabolism, 52-57
 -milk, 235-236
 in treatment of celiac disease,
 327
 of milk, 53, 158
 other than milk in infant's diet,
 56, 237, 240, 245
 overnutrition, 55
 requirement of infants, 53-55, 127
 of premature infants, 379, 388
 retention of, 55
 sensitization to, 56, 369, 371
 undernutrition, 55 (*see also* Mal-
 nutrition)
- Prothrombin of blood of newborn in-
 fant, 106-107
 of premature infant, 108
- Pseudohypertrophic muscular dys-
 trophy, vitamin E in, 109
- Pseudomonas aeruginosa as cause of
 diarrhea, 287
- Pseudoparalysis in scurvy, 456
- Ptyalin in digestion, 131
- Pulse rate in infancy, 377
 in premature infant, 377
 in scurvy, 454
- Purpura, 456
- Pyelitis, 403-405
 as cause of diarrhea, 286, 403
 of vomiting, 336
 complicating dysentery, 316-317
 malnutrition, 259, 403
- Pyloric stenosis, 340-349
 and emptying time of stomach,
 137
 as cause of constipation, 361
- Pyridoxine, 120
- Pyruvic acid, 112

R

- Rachitic rosary, 425, 426
 tetany, 430, 437, 438-449
- Rammstedt operation for pyloric stenosis, 348
- Red blood corpuscles (*see* Blood, corpuscles)
- Refeeding in pyloric stenosis, 346-347
- Reflexes in newborn, 34
- Renal rickets, 417
 failure, cause of tetany, 444, 449
- Resistant rickets, 417
- Respiration in anhydremia, 298
 in infancy, 28
 in scurvy, 454
 of newborn premature infants, 375, 376
- Rhinopharyngitis, 393-394
 as cause of diarrhea, 271, 286
 chronic, in relation to allergy, 370
 complicating malnutrition, 259
 pyelitis after, 403
- Riboflavin, 115-116
 in milk, 116, 158
 requirement, 116
- Rickets, 411-436
 acidosis in relation to, 417
 blood changes in, 423
 calcium metabolism in, 423
 celiac, 326
 congenital, 415
 diagnosis of, 431-432
 etiology of, 411-417
 growth and, 415
 incidence of infantile, 416
 in premature infants, 83, 379
 late, 417
 parathyroids in relation to, 417
 pathology of, 417-424
 phosphorus metabolism in, 423
 prevention of, 432-436
 renal, 417
 resistant, 417
 sunlight and, 411-412
 surgical correction of deformities in, 435
 -susceptible races, 411-412
 symptoms of, 424-431
 treatment of, 432-436
 ultraviolet rays in, 433
 vitamin D in, 432-434
 dosage of, 433
- Ringer's solution, 484, 485
 contraindicated with blood transfusion, 472
 in treatment of anhydremia, 293, 299
 of diarrhea, 293, 299

- Ringer's solution, in treatment—
 Cont'd
 of malnutrition, 268
 of vomiting, 355
 technique of administration, 481, 484, 485
- Roentgenograms in diagnosis of
 mastoiditis, 399
 of rickets, 429, 431
 of scurvy, 452, 453
 of sinusitis, 402
- Rosary, rachitic, 425, 426
- Rubella, maternal, effect on fetus, 19
- Rumination, as cause of vomiting, 337
- Rutin, 126

S

- Saline administration, technique of, 481, 484, 485
 in diarrhea, 293
- Salivary digestion, 131
- Salmonella enteritidis, as cause of diarrhea, 275
 in milk, 195
- Saponification of fats in intestine, 140
- Saprophytic bacteria in milk, 195
 as cause of diarrhea, 276
- Schilling hemogram, 41
 in dysentery, 315
 in malnutrition, 260
- Scopolamine hydrobromide, dosage of, 495
- Scorbutus (*see* Scurvy)
- Scurvy, 124, 450-458
 associated with celiac disease, 326
 with rickets, 457
 diagnosis of, 455-457
 etiology of, 450-451
 pathology of, 451-454
 symptoms of, 454-455
 treatment of, 457-458
- Seborrhea due to riboflavin deficiency, 115
- Second year, feeding normal child during, 248-250
- Sedatives in treatment of colic, 358
 in convulsions, 449
 in diarrhea, 309
 in dysentery, 320 (*see also* Paregoric; Phenobarbital)
- Sensitization to protein (*see* Allergy)
- Serum (*see also* Blood)
 antidysenteric, 320-321
- Shigella dysenteriae as cause of diarrhea, 275, 287
 in milk, 195
- Sight, sense of, 35

- Silver nitrate for anal fissures, 367
 for fissured nipples, 165
 not recommended in dysentery, 320
- Silver protein. color of stools after ingestion, 148
 for rhinopharyngitis, 394
- Sinusitis, 402-403
 as cause of diarrhea, 308, 402
- Skeletal age, 31
 muscle, 36
- Skeleton, growth of, 30-31 (*see also* Bone)
 mineralization of, 82
- Skin, development of, 32
 in anhydremia, 298
 in malnutrition, 258, 259
- "Skyskine" in relation to rickets, 412
- Smell, sense of, 35
- Soap stick for constipation, 366
- Soaps in stools, 140, 148
- Sodium bicarbonate, solution for intravenous use, 484
 bromide, dosage of, 495
 chloride (*see* Physiological salt solution)
 citrate, dosage of, 495
 for blood transfusion, 469, 470
 lactate, 300-301
 dosage of, 495
 for parenteral administration, 484
 metabolism, 77, 78-82
- Solutions for intravenous use, 484, 485 (*see also* Dextrose; Ringer's solution; Physiological salt solution)
- Soybean in infant feeding, 56, 237, 372
- Spasmophilia (*see* Tetany)
- Spasticity, 33, 118
- Special foods in infant feeding, 229-237
- Specific dynamic action, 47
- Spleen in rickets, 424
- Staphylococci as cause of diarrhea, 276
 of mastoiditis, 398
 in gastrointestinal tract, 141
 in urinary cultures, 461
- Starch, contraindicated in celiac disease, 327
 hydrolysis of, 63
 in infant feeding, 68
 dextrinized, 63, 64
 in stools, 149
- Starvation, as cause of diarrhea, 252, 261
 period in treatment of diarrhea, 291, 305
- Steapsin (lipase), 135, 139
- Stenosis, pyloric (*see* Pyloric stenosis)
- Stillbirth, incidence of, 18
- Stomach, absorption from, 136
 capacity of, 29
 development of, 28-29
 digestion in, 132-137
 emptying time of, 136
 overdistention of, as cause of vomiting, 334
- Stomatitis, ulcerative, in scurvy, 455
- Stools, 145-151 (*see also* Constipation, Diarrhea, Dysentery)
 bacteria of, 150-151, 288
 blood in, 149
 cultures, technique of, 461-462
 examination, significance of, 151
 in celiac disease, 323, 325
 in constipation, 361-363
 in diarrhea, 278
 in dysentery, 314
 in intussusception, 351
 in mastoiditis, chronic, 400
 in pancreatic fibrosis, 331
- Streptococci, hemolytic, as cause of enteritis, 275
 of mastoiditis, 398
 growth inhibited by gastric acidity, 141
 in gastrointestinal tract, 141
 in milk, 195
 in urine cultures, 461
- Streptococcus fecalis, in gastrointestinal tract, 141, 142, 143
 in milk, 195
- Streptococcus lactis in milk, 195
- Streptomycin, dosage of, 495
- Stridor, in tetany, 442, 444
- Strong, bacillus of, 312
- Subcutaneous tissues, growth of, 32
 injection, 481-482, 484, 485
- Subperiosteal hemorrhage in scurvy, 454, 455
- Succinyl sulfathiazole in dysentery, 321, 495
- Succus entericus, 137
- Sucrose in infant feeding, 59, 63, 70
- Sugar (*see also* Carbohydrate or name of sugar)
 as cause of diarrhea, 69
 cane (sucrose), 59, 63, 70
 in prophylaxis of diarrhea, 289
 in urine, 59
 laxative effect of, in constipation, 70, 364

Sugar—Cont'd

- malt, 63
- of blood, 59, 60
- Sulfaguanidine in treatment of dysentery, 321
- Sulfapyrazine, 321
- Sulfonamides, 321
 - and paraminobenzoic acid, 122
 - dosage of, 495
 - in treatment of pyelitis, 404
- Sulfur, 77, 86-87
- Sulfur-containing amino acids, 158, 159
- Sunlight in relation to rickets, 411-412
- Superficial reflexes in newborn, 34
- Supplemental feedings, 175
- Suppositories, use of, in constipation, 366
- Surgical treatment of pyloric stenosis, 348-349
- Swallowing of air as cause of vomiting, 333
- Sweating, head, excessive in rickets, 430
- Sweet milk mixtures, 203-212
- Sweetened condensed milk, 202, 232
- Syncope in mastoiditis, 401
- Syphilis, congenital, 407-410
 - no contraindication to breast feeding, 155, 410
 - osteochondritis of, differences from scurvy, 456
 - treatment of, 408-410

T

- Technique of administration of oxygen, 385-386
 - of salt solutions, 481, 484, 485
- blood collection, 462-468
- blood grouping and matching, 468-469
- blood transfusion, 469-481
- dextrose administration, 483-485
- gavage, 487-488
- intraperitoneal injection, 482-483
- intravenous injection, 469-487
 - continuous, 485-487
- stool culture, 461-462
- urine collection, 459-461
- urine culture, 461
- venoclysis, continuous, 485-487
- Teeth affected by rickets, 430
 - by scurvy, 455
- development of, 26-27
- vitamin D and, 104
- fluorine and, 92-93
- Temperature (*see also* Fever)
 - of infant with anhydremia, 283, 297

Temperature, of infant—Cont'd

- with dysentery, 314
- with malnutrition, 260
- of newborn infant, 36
- of nursery for premature infant, 383
- regulation of, of premature infant, 378
- Tendon reflexes in newborn, 34
- Tetany, 437-449
 - age, effect of, on symptoms, 443
 - as effect of vomiting, 354
 - associated with celiac disease, 326
 - with pyloric stenosis, 342
 - with rickets, 430, 439
 - blood changes in, 84, 437, 440, 444
 - diagnosis of, 443-445
 - etiology of, 437-440
 - neonatal, 437
 - pathogenesis of, 437-440
 - symptoms of, 440-443
 - treatment of, 445-449
- Theobromine sodiosalicylate (diuretin), dosage of, 495
- Thermolability of premature infant, 378, 381-384
- Thiochrome, 111
- Thiamine, 111-115
 - in milk, 113, 158
 - in relation to constipation, 361, 365
- Thickened feedings for pyloric stenosis, 344, 346
 - for rumination, 337
- Thorax, growth of, 27
 - in rickets, 425, 426, 427
- Thymus, 33, 42
- Thyroid and bone metabolism, 82
 - dosage of, 495
 - deficiency, 256, 361, 415
 - effect of iodine deficiency on, 91
 - weight of, at birth, 91
- Thyroxin, 91
- Tissues, subcutaneous, growth of, 32
- Tomato juice as source of vitamin C, 239, 457
- Tonsillitis accompanying otitis media, 397
 - accompanying pyelitis, 405
 - accompanying rickets, 424
- Touch, sense of, 35
- Toxemia as cause of vomiting, 352
 - from bacteria, 280, 285
 - in dysentery, 314
- Toxicosis, 298 (*see also* Diarrhea)
- Transfusion, 469-482 (*see also* Blood transfusion)
- Tridione, dosage of, 495
- Trousseau's sign in tetany, 441, 443
- Trypsin, in digestion, 137

- Tryptophane, absence of, in gelatin, 52
and nicotinic acid, 118
- Tube feeding for premature infant, 389, 390
- Tubercle bacillus in gastrointestinal tract, 141
- Tuberculosis, 406-407
as cause of malnutrition, 255, 407
of middle ear, 397
of mother, contraindication to breast feeding, 154, 406
- Twilight blindness as symptom of riboflavin deficiency, 115
- Typhoid bacillus in gastrointestinal tract, 141
in mother, contraindication to breast feeding, 155
in stool cultures, 461
in urine cultures, 461
- U
- Ulcers in dysentery, 313, 315
- Ultraviolet rays (*see also* Vitamin D)
and etiology of rickets, 412
in treatment of rickets, 433
- Umbilical hernia in rickets, 428
- Underfeeding (*see* Malnutrition)
- Undernutrition (*see also* Malnutrition)
of protein, 55
- Underweight infants, requirements of, 127
- Urinary tract (*see also* Pyelitis)
growth of, 30
- Urine, collection technique, 459-461
cultures, technique of, 461
in anhydremia, 284
in dysentery, 315
in malnutrition, 260
in pyloric stenosis, 342
in scurvy, 455
in newborn, 30, 79
in premature, 79, 380
- Urograms, in pyelitis, 405
- Urotropin (Methenamine), dosage of, 495
- Urticaria in relation to allergy, 370
- Utilization of food, deficient, as cause of malnutrition, 254
effect of infection on, 254
- V
- Vaccines not valuable in treatment of dysentery, 321
- Vegetable protein in infant feeding, 56, 237, 372
- Vegetables in infant feeding, 242, 247-250
in relation to constipation, 365
- Veins, conservation of, 468
- Venipuncture, technique of, 464-466
- Venoclysis continuous, 485 (*see also* Intravenous therapy)
- Vernix caseosa, 100
- Vineent's angina relieved by treatment with nicotinic acid, 119
- Vioosterol (*see* Vitamin D)
- Visual purple, 96
violet, 96
- Vitamin A, 96-102
for premature, 392
in milk, 158
requirements for, 100, 130
- Vitamin A₂, 96
- Vitamin B complex, 110-122 (*see also* separate vitamins)
and respiratory enzymes, 110
components of, 110
biotin, 120
choline, 121
folic acid, 119
inositol, 121
niacin, 117-119
pantothenic acid, 121
paraminobenzoic acid, 121
pyridoxine, 120
riboflavin, 115-116
thiamine, 111-115
content of milk, 158
deficiency, multiple, 111
requirements for, 130
- Vitamin B₁ (*see* Thiamine)
- Vitamin B₂ (*see* Riboflavin)
- Vitamin B₆, 119
- Vitamin C (*see* Ascorbic acid), 122-126
- Vitamin D, 102-106
as supplement for breast-fed infant, 176
concentrates of, 103, 433
deficiency, 102, 411-436
dosage in rickets, 433-434
in formation of bone salts, 82
in rickets prevention, 104, 411-412, 432
in premature infants, 379, 391, 434
in treatment of rickets, 432-434
of tetany, 445
intramuscular administration of, 434
massive doses of, 105, 434, 435
milk, 201, 215
occurrence of, 103, 411
requirement of, 103-106, 129

- Vitamin D, requirement of—Cont'd
 for skeletal growth, 103
 of premature infant, 391, 434
 Vitamin D₂, 103
 Vitamin D₃, 103
 Vitamin E, 109-111
 Vitamin G (*see* Riboflavin)
 Vitamin H, 120
 Vitamin K, 106-109
 dosage of, 495
 Vitamin M, 119
 Vitamin P, 126, 130
 Vitamin W, 120
 Vitamin, pellagra-preventive (*see*
 Nicotinic acid)
 Vitamins, 95-126, 129-130
 deficiencies of, multiple nature of,
 111
 in evaporated milk' (*see* Evaporat-
 ed milk)
 in milk, 158
 Vomiting, 333-355
 alkalosis in, 341, 354
 and absorption of vitamin K, 109
 blood findings during, 354
 caused by air swallowing, 333
 by allergy, 352, 370
 by anhydremia, 352, 391
 by atresias, 339, 349
 by gastroenterospasm, 338
 by habit, 337-338
 by histamine, 274
 by improper clothing, 336
 by intestinal obstruction, 338-
 339, 349, 350
 by overdistention of stomach,
 334-335
 by parenteral infections, 336
 by pyelitis, 403
 by pyloric stenosis, 340-349
 by rumination, 337
 by toxic states, 352
 by unsuitable food, 335-336
 convulsions and, 354
 cyclic, 352
 effects of, on body, 353-355
 Vomiting—Cont'd
 in diarrhea, 284, 298
 in malnutrition, 261, 265
 in mastoiditis, 398, 400
 in otitis media, 394
 in premature infant, 387, 390
 in rhinopharyngitis, 394
 projectile, in pyloric stenosis, 340
- W
- Water (*see also* Anhydremia; Dehy-
 dration; Edema)
 absorption of, from intestine, 141
 balance in dysentery, 318
 extracellular, definition of, 78
 interstitial, definition of, 78
 intracellular, definition of, 78
 labile, 79
 loss in diarrhea, 281
 metabolism of, 78-82
 need of, in diarrhea, 299
 requirement of infant, 78-82, 129
 Weaning from breast, 177
 temporary, 155
 Weight, body, 22, 171, 384
 charts, 20, 21
 Well-water cyanosis, 188
 Wet nursing, 178
 Whey protein (*see* Lactalbumin)
 White blood corpuscles (*see* Blood
 corpuscles, white; Hemo-
 gram, Schilling)
 Whole sweet milk feeding of nor-
 mal infant, 203-212 (*see*
 also Feeding, artificial)
- X
- Xerophthalmia in vitamin A defi-
 ciency, 97
 X-ray, (*see* Roentgenograms)
- Z
- Zinc, metabolism of, 93-94



charlie
26/6/13

~~100~~ 6.10.00

24/8/89

~~100~~ 5.5.97



CFTRI-MYSORE



2540

Infant nutrition.

